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HYDATIDIFORM MOLE

A Plea for the More Careful Study of Tissue from Abortuses

Report of Three Cases

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(Read before the thirty-ninth session of the Arizona State Medical Association, held in Phoenix, Arizona, April 24 to 26, 1930).

"To read the titles of articles on 'molar' pregnancies which have appeared during the last few decades, even, is a rather wearisome task. By far the great majority of the articles concern themselves merely with the report of 'a case' or (rarely) of 'several cases' of hydatidiform moles. The recent cancer literature stands in marked contrast to this, for not even the general practitioner would think of reporting a routine case of cancer of the breast, let us say. The significance of these fact is self-evident, and whatever else they may mean they do imply that hydatiform mole still is regarded as a rare condition. Indeed many of those reporting 'a case' frankly say so, and although the incidence of hydatiform degeneration is estimated variously by different authors and investigators, there seems to be entire agreement that it is a rare, even if not an extremely rare, condition. This opinion seems to be shared even by those general practitioners whose long practice runs high up into the hundreds or even into the thousands of obstetrical cases. Indeed, many general practitioners declare that they have not seen a single case of hydatiform mole during the practice of a long life." (Meyer).

Had I been familiar with the above paragraph quoted from the classic contribution of Arthur William Meyer, published by the Carnegie Institute of Washington, I never would have had the temerity to add this paper to an all too prolific literature. While I stand indicted on every count in the introductory paragraph, I am too deeply committed to retract, having already presented the subject to the Yavapai County Medical Society and having had it accepted for this meeting.

I now offer the following reasons for calling your attention to the subject of hydatidiform mole:

1. It introduces a pathologic entity which has not been discussed before any of the Southwestern Associations in the last thirteen years.

2. Because its history abounds in interesting speculations, discussions, and contradictions as to etiology and pathology.

3. Because the vagaries and contradictions concerning the frequency of its appearance furnish instruction in statistical evaluation.

4. Because, after thirty-three years of "watchful waiting," I witness my first case, to be followed by the recognition of two immature forms within nine weeks.

5. Because I should like to record these cases and review with you some of the factors in the modern treatment of the condition, which are so at variance with the teachings of three decades ago.

6. Because I sincerely desire to create an interest in the study of tissue from abortuses (spontaneous) by a simple method I shall describe, hoping thereby to find more immature forms of hydatidiform mole and thus increase the value of our statistics and the accuracy of our prognoses.

Synonyms: Hydatidiform mole is known also as vesicular mole, myxoma chorii, cystic degeneration of the chorion, grape mole and chorioma benignum.

Definition: Hydatidiform mole is a disease associated with pregnancy, in which the chorionic villi are deformed in structure. The placenta fails to form a compact mass, its villi branching loosely in clusters. The stalk, or stoma, of the villus becomes edematous and distorted, and in the majority of cases the covering layer (the Langhans cells and syncytium) proliferates unduly.

The name hydatidiform originated because of the close resemblance to echinococcus cysts (Brenser 1819). R. R. Frank, S. H. Geist, Dean Lewis' Surgery.

Historical: A number of writers state that Hippocrates knew of this condition. Kessman states that Hippocrates ascribed the cause to a mucoid degeneration of the villi.

Apparently there is a lapse of nearly ten centuries before the subject is again definitely presented in the literature. Aetius of Amada, writing early in the sixth century, clearly described it. Again a lapse of about ten centuries. Vega in 1564 and Valleriola in 1573 reported typical cases. Doubtless due to the uncanny appearance and prolific numbers of these "cystic ova" and the mystery which then shrouded their origin, the literature became large and difference of opinion rife.

Valleriola believed the disease due to the abnormal development of the "female semen." Regnier de Graf (1678) thought that each vesicle was an unfecundated ovum. We have definite proof that prior to 1223 the opinion prevailed that each cyst was a living embryo, and thereby hangs a tale, a bit of medical lore concerning "Black Meg," handed down to us through the writings of the great surgeon Ambrose Pare and Samuel Pepys, to whom nothing was sacred, not even his thoughts, as his diary recounts. Pare details the case of the Countess Margaret of Flanders, "Black Margaret," daughter of Baldwin, Emperor of Constantinople, who brought forth at one birth 365 infants, of whom 162 were solemnly baptized "John," 182 "Elizabeth" and the odd one, after much ecclesiastical debate, was adjudged an hermaphrodite and accordingly buried without baptism. Pepys in his journey to the Hague in 1660, speaks of going by wagon to Lansdune, where the 365 children were born: "We saw the hill where they say the house stood and sunk, wherein the children were born, the basins wherein the male and female children were baptized do stand over a large table that hangs upon a wall, with the whole story in Dutch and Latin, beginning Margaret Herman Comitissa."

Countess Margaret was granted a divorce from her first husband, Bosschaert (Buchard), Lord of Avennes, on several grounds, one of which was "too near relationship between the parties." My historic link is broken at this point but, assuming Bosschaert responsible for the pregnancy resulting in this mole of 365 children, we can readily understand from the beliefs prevalent at the time why the grounds for divorcement were sufficient.

Paul Postal (1685) presented in his work a good picture of the condition, with a fairly accurate description. Ruysch in 1691 declared that the cysts were due to some disease or alteration of the ovum. In the early part of the nineteenth century Madame Boivin, a French midwife, published several papers on obstetric subjects. She is credited by one author as considering the disease due to echinococci, by another as recognizing the chorionic origin of the mass. Velpau (1827) called the cysts degenerative villi, as we know them today. Meckel (1847) suggested the hydrops due to vascular destruction. Virchow (1853) called it a mucoid myxomatous degeneration, a view generally accepted until 1875 when it was disproven by the masterly study of Marchand.

With the first report of chorion epithelioma by Sanger in 1889 and the rapidly confirmed observation that more than one-half of such cases were preceded by the expulsion of a hydatid mole, great interest was created in the pathology of the two conditions, especially the question as to whether mole was an essential precursor to the development of chorion epithelioma. In 1922 Emil Novak stated that no group of neoplasms is more interesting than that represented by hydatidiform mole and chorioepithelioma. Furthermore, DeLee states these malignant syncytiomata may develop in the uterus, in the cervix, or in the vagina (local metastases), or, if the vesicles and masses of syncytium are carried by the blood stream to distant organs, a general syncytiomatosis may ensue, but usually the lungs are first and most involved.

Frequency: Statistics on the frequency of hydatidiform mole exhibit such wide variations that we welcome any plausible explanation. It seems reasonable, as pointed out by Schumann, that many cases of this affection occur in the hands of practitioners outside of hospitals and are never reported, that a true evaluation of their frequency is not possible. Another noteworthy observation by Meyer is that "mole formation is a rare disease at or near term, but it probably is the commonest of all diseases of the ovum during the earliest months of gestation. The typical large hydatidiform mole is an end result which has taken many months to develop." My own observation from a study of the literature is that there will always be, and justly so, a wide margin between statistical evaluations of the surgeon based on macroscopic appearance, and those of the pathologist based on microscopic study. Having oriented ourselves upon the cause of these variations in the statistical incidence, the following estimates become the

more interesting. Essen-Moller gives the incidence as three per thousand, while Madam Boivin considered it far more rare, occurring only once in 20,000 cases. Williamson gives an incidence of one in 2,400 cases. Pazzi is quoted as having stated that he had never, in an experience of more than 6,000 cases, encountered a case of hydatidiform mole. Edgar stated, "I have seen it (hydatidiform mole) four times in 15,000 cases of labor observed in hospital and in private practice."

Meyer calls attention to the overlooked investigations of Gierse, in 1847, who found that the characteristic changes of hydatidiform disease are found in many abortions and, indeed, constitute a most common cause of abortion in the early months. Gierse believes that hydatidiform degeneration is an extremely common condition in spite of the rarity of the fullblown form of the disease, to which authors have usually confined their attention. Storch, in 1878, likewise emphasized the frequency of hydatidiform disease in early pregnancy. Meyer urges that the cases of hydatidiform degeneration reported in the literature are merely end results of a process which, in its early stages, is a common cause of early fetal death. He estimates the incidence of these earlier forms of the disease among the fetuses in the Mall collection, including some of the specimens classed as normal, as 10.9 per cent among the uterine specimens and 20.8 per cent among the tubal. He estimates, tentatively, that as many as from four to ten per cent of all conceptions end in hydatidiform degeneration. These figures are rather startling, for they indicate an incidence fully 240 times as great as that given by Williamson, and 33.3 times as great as that given by Essen-Moller (Novak).

According to Findly, molar pregnancies occur once in 3,000 normal gestations. Schumann, summarizing his discussion, states "that for practical purposes the incidence of one (hydatidiform mole) in about 2,000 pregnancies seems to portray the average frequency. Kaufmann, quoting Seitz, states that hydatidiform mole occurs once in 500 cases of childbirth.

In relation to abortuses, O. A. Gordon, Jr., makes the following observation from the Bellevue Hospital records: "Out of approximately 4,500 abortions we have discovered twenty-one hydatidiform moles, or 0.4 per cent. I say we have discovered twenty-one cases, for I feel sure many early cases have been overlooked."

Hydatidiform mole is more frequent in multipara and during the middle of the childbearing period. From Findly's table we note that between fifteen and twenty-five years there were 111 cases; between twenty-five and thirty-five years of age, 143; between thirty-five and forty-five years of age, eighty-four; and between forty-five and fifty years of age, thirty-six cases. These figures probably do not bear a normal ratio to the total pregnancies for these years, certainly their frequency after twenty-five is the more remarkable when we consider the small number of births at this period.

Ectopic gestation may undergo this form of degeneration but hydatidiform mole of the tube may be considered rare. Novak states "that in a number of tubal specimens which I have recently examined, however, there seems to be no question as to the character of the villous changes." In view of this statement the figures from the Mall collection of 20.8 per cent of mole in tubal pregnancy does not seem absurd, but rather suggests that the surgeon is not submitting his specimens of chorionic tissue to the careful pathological study they deserve.

Repetition of molar pregnancy in the same individual is rather rare, 1.4 per cent, using Findly's figures. Essen-Moller reported the case of a woman who expelled spontaneously eighteen moles in the course of nine years. I record in this report the detailed history of a case (Case 3) who had four moles in twelve pregnancies.

Appearance and Size: We should remember that the appearance and size varies, with the period of development, from a few tiny vesicles in an aborted ovum to the fully developed cysts seen in later months.

DeLee states that hydatidiform mole (mature) can not be mistaken. It resembles a bunch of catawba grapes of most irregular size, varying from that of a pin-head to that of a hen's egg (rare), but usually the size of a pea or bean. Years ago Playfair stated that, on minute examination, they are found not to be each attached to independent pedicles, as is the case in a bunch of grapes, but some of them grow from other vesicles, while others have distinct pedicles attached to the chorion. The pedicles themselves sometimes are distended with fluid.

A study of my own fully developed case shows each cyst attached, (1) by a delicate, sometimes threadlike pedicle to each other, (2) to parent cysts evincing a process of budding not unlike that observed in yeast, and (3) to dense areas of decidua.

A well developed mole weighs from 300 to 600 grams, a weight of 8000 grams has been recorded (Menu). Lusk relates his experience with one which filled an ordinary wooden bucket. In this case there had been se-

vere hemorrhage, the patient dying two hours after delivery from anemia and shock.

Etiology: The etiology is uncertain and much in dispute (Kaufmann). Frequent association with nephritis has been noted and Durante believes that maternal toxins are a cause. Syphilis, heart disease, general affections, and disease of the ovum and spermatazoon have all had their advocates, but they are practically ruled out today as having no demonstrable foundation. According to Meyer, "of the many explanations which have been offered for the advent of hydatidiform degeneration, none seems to be better established than that of endometritis."

After reading a great number of theories and opinions, we quote Schumann: "It is sufficient to say that with our present knowledge of the subject, the etiology of mole formation is not known, but the evidence seems to point to . . . . changes taking place in the embryonal envelope rather than in the embyro itself."

Hydatidiform Degeneration in Other Mammals: I had hoped to approach the etiology of hydatidiform mole through the study of comparative anatomy and pathology. So far the results have been disappointing. DeLee states that Aichel had exposed the uteri of pregnant dogs, injured the placental site by pressure and observed the formation of hydatidiform mole in seven to thirteen experiments. Professor Meyer, commenting on this experimental "evidence," states: "Although a condition closely simulating hydatidiform degeneration may be produced experimentally by pressure, I doubt that pressure is the cause. There are so many facts which would seem to be irreconcilable with such a hypothesis."

From the library of the Surgeon General I secured abstracts of two references kindly furnished by Dr. Buckley, Chief Pathological Division, Bureau of Animal Husbandry, Department of Agriculture, Washington. C. Wililams of Ithica, New York, has reported cases but his observations are questioned by Joest (Berlin) thus: "The formations described by Williams do not correspond actually to what is termed hydatid mole, according to accepted definition. The hypertrophy of the villi is absent. The formation is nothing else but an ovum, the embryo of which has perished, and the membranes of which have continued to grow. The hypertrophy and edematous or myxomatous degeneration of the villi are especially absent."

Frei (published by Joest) describes a case from a cow, with photograph showing numerous cluster-like and cystic formations ranging in size from a pea to a child's head. Grossly this might be chorionic degeneration but the histologic and pathologic data are hardly sufficient to classify it as of the same type of chorionic cystic degeneration observed in the human. Frei gives a bibliography which I will append in its proper place.

Lacking full histologic and pathologic confirmation I will quote Professor Meyer as representing the best present American views on this point: "At the time I was engaged in my investigations on these matters, I wrote to the foremost veterinarians in colleges connected with universities, but did not succeed in learning of a single authentic case of hydatidiform degeneration in other mammals. One or two men wrote saying that they had heard of it, but reference to the literature always left me without confirmatory evidence. We have never observed it in the many thousands of laboratory animals—rabbits, guinea-pigs, rats, mice—which we have used during my presence here at Stanford."

Pathology To promote clarity in considering the pathology of mole, we will pause to review briefly the histology of the epithelium of the normal villi. There are two layers; the lower, Langhan's, is made up of clear cuboidal cells, undoubtedly of fetal origin, trophoblastic. An outer protoplasmic layer or syncytium is rich in darkly staining nuclei, containing fat granules and a striated border. There are two opniions as to its origin: one, that it is maternal, representing changed uterine epithelium: second, it is of fetal origin. The majority of workers believe both layers arise from the primary ectoderm, the trophoblast of the germinal vesicle. (Kaufman).

The three pathologic changes which characterize hydatidiform mole are trophoblastic proliferation, hydropic degeneration of the stoma and degeneration of the blood vessels (Novak). Furthermore, it should be remembered that different pictures are likely to be encountered in different parts of the mole and that trophoblastic proliferation may be striking in one area and absent elsewhere. These peculiarities are perhaps chiefly due to the environmental conditions surrounding the villi, depending upon whether the villus has separated from the uterine wall or whether it still receives abundant nutrition from the maternal tissues. (Novak.)

Each individual cyst is a swollen and edematous chorionic villus, its entire covering being the ectoderm as more or less proliferated Langhan's cells, exuberant and irregular buds of syncytium, and a delicate stem. (Schumann.)

The hydropic and partially mucoid changes beginning in the center of the stoma and leading to bubble-like blowing up of villi are not the most important part of the disease; the changes begin rather in the epithelium of the villi where there is a vigorous more or less lawless overgrowth with mitoses, chiefly on the part of Langhan's cell layers. (Marchand-Frankel.))

The mole process may involve the entire chorion, since originally this contains villi over all of its surface, and then no placenta can be recognized, or the process may involve the placenta alone, or only isolated cotyledons. The last is quite common in abortion and is important in considering chorion epithelioma. If those changes take place in the first two months of pregnancy, the entire ovum is changed into a mole, and there can be found no amniotic sac, no fetus and no placenta (sterile mole). (Kaufmann.)

Relation to chorio-epithelioma: When the fact became known (Sanger 1889) that the disease (hydatidiform mole) might be either benign or malignant, every effort was made by pathologists to discover some distinguishing histologic mark by which the two forms might be differentiated. These efforts have been unsuccessful. Marchand, Aschoff, Zagorianski, Kissel, v. Franque, Albrecht, Hormann, and others agree that we have absolutely no histologic criterion for determining the malignancy of these tumors. Moreover, a further difficulty in diagnosis exists in the fact that the tumors exactly resemble the normal fetal tissue. Hitschmann and Cristofoletti came to the following conclusion: "As a matter of fact, we are convinced that a histologic difference is entirely improbable. We know of absolutely no morphologic and biologic difference between the **physiologic** fetal tissue and the **pathologic.** The more carefully we study tbe earliest phases of placenta formation and compare them with chorio-epithelioma, the more we feel justified in the statement that between **trophoblast** (fetal tissue) and **chorio-epithelioma there is no difference.** (Graves.)

Diagnosis and Symptomatology: Hydatidiform mole occurs most frequently in multipara and in the middle of the childbearing period. The diagnosis is often not made until grape-like cysts have been extruded and preserved for the physician's inspection. Failure to literally "drag out" this fact from the patient or nurse in your history taking may result in your failure to make a correct pre-operative diagnosis, as in my first case where two "placental" masses were exhibited but two cysts as large as grapes were neither exhibited nor mentioned until after operation.

Given a history of suppressed menstruation, followed in two or three months by repeated, intermittent or prolonged mild or severe hemorrhage, without known cause, often unaccompanied by uterine pains, and then a careful bimanual examination revealing an enlarged, soft or elastic (not doughy) uterus, somewhat larger than the period of gestation would suggest, should at least arouse suspicion as to the presence of hydatidiform mole. If the cervix is patulous and the cysts of the mole palpated, the diagnosis becomes positive. Extruded cysts are pathognomonic. In later months the disproportionately enlarged uterus, without detection of fetal heart sounds or motion, might raise the question of mole. On the other hand, we would not overlook the fact that a well developed mole and a living fetus have been reported to co-exist in the uterus, but it is extremely rare and is explained either by a twin pregnancy or only a small portion of the placenta involved in mole formation. Another point to be developed in the history at least, is that many of these women are "sick," run down, toxic or anemic out of all proportion to the hyperemesis or bleeding experienced.

In the bimanual examination we are statistically informed that fifty per cent of the mole cases show the enlarged bilateral lutein cysts of the ovaries. DeLee states that in two of his cases the uterus became smaller, not larger, before the mole was expelled. This fact was noted in two of my cases. In the abortion itself the uterine contractions are usually sluggish but the hemorrhage is often alarming and furious, and death may ensue from acute anemia.

In differential diagnoses we have to consider the ordinary abortion, hydramnios, placenta previa and grape-like sarcoma of the cervix.

Prognosis: Fetus: The fetus is usually destroyed early during the mole growth and is seldom found when the mole is discharged.

Maternal: This is of importance, first, as to the immediate effect from hemorrhage, sepsis (peritonitis) and rupture of the uterus, these factors being further modified by the age of the mole (immature and full-blown); second, somewhat more remotely, we are concerned with the invasive, malignant type, chorion epithelioma.

Here again statistics are at variance. A primary mortality of ten and fifteen per cent is probably fairly accurate if cases of Class 2 are excluded. In Class 2 (chorion epithelioma) the mortality immediately becomes enormous (fifty per cent—Schumann). We note, "That in a carefully observed series from five to sixteen per cent of hydatidiform moles are followed within

five years by true chorion epithelioma" (Schueitzer, Sunde, Essen-Moller). The danger of chorion epithelioma following the development of hydatidiform mole is increased as the age of the patient advances; in those over forty-five years of age the danger is five times as great as in a younger patient (Frank and Geist).

Treatment: We might briefly summarize the treatment of thirty-five years ago as "non-interference so long as the uterus remained passive. When contractions set in the vagina was tamponed and ergot given in full and repeated doses until the mole was expelled, or rapid dilatation of the cervix and manual removal of the mole, after which the uterus was washed out with antiseptic fluids or swabbed out with perchloride of iron."

Today we find treatment sponsored by two schools which we shall call conservative and radical. The practice in each, however, is often modified by the stage of development of the mole and the ravages of hemorrhage and sepsis encountered. Furthermore, from what has been stated concerning pathology and prognosis, these two schools base their differences in treatment very largely upon their evaluations of these two factors.

The conservative school would ordinarily empty the uterus from below. Here adequate dilatation of the cervix is imperative. Furious hemorrhage may be encountered at the first attempt at dilatation and gauze pack may have to be resorted to, resulting at times in a two stage operation. Rigid asepsis is imperative and the work should be done in light anesthesia, but many of these cases are "emergencies" outside of hospitals. If hemorrhage is not too furious, or the patient's depletion too great, cervical dilatation to admit one or two fingers is highly desirable, not only because it permits a more accurate and rapid removal of the mole but because digital exploration of the uterine cavity during and after removal of the mole is necessary to the safety of the patient and your prognosis. In emptying the uterus from below we can not overemphasize the meticulous care required in digital and instrumental removal of the mole. The tactus eruditus seldom serves a greater need. Perforations are common and hemorrhage often alarming. One or both of these factors may force the conservative operator to pursue the radical course, open the abdomen and perform a hysterectomy even before a much needed transfusion can be done.

The conservative treatment is summarized by O. A. Gordon, Jr., as follows: "Hydatidiform mole should be treated from the standpoint of hemorrhage and sepsis, bearing in mind only the remote possibility of the development of chorio-epithelioma."

Having explored the uterus carefully with the finger (if possible) and removed all mole tissue by digital curettage supplemented by the use of one of the least traumatizing types of placental forceps, we swab the uterus gently but thoroughly with a gauze wipe carrying half strength tincture of iodine, followed by a dry gauze wipe and then followed by an iodine gauze pack for twenty-four hours. To overcome the uterine inertia, so often noted in these cases, the hypodermic administration of aseptic ergot or small doses of pituitrin is advised.

Schumann advises against the use of the curette and placental forceps whenever possible because of the danger of perforation by this means, and because of the danger of distant metastases, as to the lung, being produced by instrumental removal.

The diganosis made, the radical school would proceed at once to open the abdomen and do either a supravaginal hysterectomy or cesarian section. If the patient has been adequately compensated for blood lost, by transfusion with salines, with the operation skilfully and rapidly performed under ideal operating conditions, either of these radical procedures has at least two factors to its credit: (1) The hemorrhage is almost immediately controlled in the steps of the hysterectomy or by manual compression of vessels in the cesarian section; (2) there can be no incomplete removal of the mole or possibility of chorion epithelioma later.

If the uterus is opened and the mole removed, it is done in the "open" with the cavity of the uterus under optical and digital inspection. Should the syncytium be observed to have penetrated deeply the uterine wall, hysterectomy can complete the operation. If after removal of the mole, inspection reveals no serious damage to the uterine mucosa, the uterine musculature may be closed as in cesarian section, having first passed a strip of iodoform gauze down through the cervix into the vagina.

That the lutein ovarian cysts so frequently associated with hydatidiform mole can be promptly dealt with in the abdominal operations is hardly a valid argument for the major procedure because as previously noted, many cysts involute after the mole is expelled and cause no further trouble. Extensive radical surgery in these poor risk cases as a routine would certainly add to the immediate mortality. The desirability of leaving the uterus should always receive consideration in selecting the operation. Many mole bearing uteri have subsequently

brought forth living healthy children, as illustrated by two of my cases.

Finally, as noted elsewhere in this discussion, many of these cases occur in the hands of practitioners outside of hospitals. Here the resourcefulness of the physician is taxed to the utmost. He must stop a furious hemorrhage rapidly attaining alarming proportions and, at the same time, prevent sepsis by the use of such emergency material, instruments and appurtenances as are available, attended often by untrained assistants and without anesthetic. These are the situations that frequently add to the mortality statistics.

The severity of the hemorrhage is not always in proportion to the size and age of the mole. Early moles showing only minute degeneration in very young villi may open up large intervillous spaces or uterine sinuses, causing prolonged bleeding, followed by severe hemorrhage until the cause is removed.

Postoperative observation should be had in all cases of hydatidiform mole for several months and a follow-up record for a period of at least five years. Without causing undue apprehension, I believe the patient should be instructed concerning the general factors for health after hydatidiform mole and her intelligent cooperation sought. More particularly is this true as she passes into the hands of other physicians.

Examination of Tissues from Abortions: I have found the following simple method of service in the gross study of the aborted products of conception.

In cases of irregular bleeding ("threatened abortions") in the early months of pregnancy, or even where patients are passing "fleshy" tissue with the blood clots, I provide them with a small wide-mouthed specimen bottle containing several ounces of two per cent formaldehyde solution. They are instructed to separate the clots from the "fleshy" particles and send the latter in at the first opportunity. Should the specimen bottle not be large enough they are directed to use a fruit jar or other similar receptacle, to wash the blood clots out gently and put the "fleshy" portions in the jar, covering with enough water to prevent dehydration, which is rapid in this climate.

On receiving the specimen I "rinse" it every two or three hours in two per cent formaldehyde solution for the purpose of removing blood and slightly hardening the delicate structures.

The specimen, or protion of it, is now transferred to a small wide-mouthed flat (not round) bottle or specimen jar containing two per cent formaldehyde solution and the container stoppered. With Leupe glasses or hand magnifying glass, interpose the specimen between the eye and a strong light, gently revolve or agitate the specimen in the clear solution. A very young hydatidiform mole will be easily detected, its syncytial buds floating like kelp (sea weed), which it greatly resembles at this stage. Its recognition when a vilus becomes hydatiform, that is, when liquifaction of the stoma occurs. "This liquifaction appears in more or less restricted portions of the villus, thus giving rise to the long fusiform and later spherical vesicles so characteristic of the hydatiform mole" (Meyer). The typical gross, hydatid or watery, translucent nature of the villi can not be relied upon in early stages, for normally shaped villi, which have undergone considerable lysis may be almost transparent and also somewhat more than normally bulbous. However, save in the case of some specimens of tubal pregnancy, the swelling of the villi, due to maceratioin or to luetic changes, is quite different in character from that characteristic of hydatiform degeneration, and usually quite easily distinguishable from it."

A further refinement of the gross study may be made by grasping with fine tissue forceps, under direct vision the particular cluster of villi desired and lifting it out of the solution (it will usually sever by its own weight, if not, cut with fine scissors); place in a hanging drop slide, filling the cup with five per cent formaldehyde solution; spread the villi by teasing with a fine needle; make a moat of Canada balsam and apply a cover glass. Examine under very low power of the microscope, four to twenty diameters. A microphotograph can readily be made at this time if desired. Confirmation of the macroscopic findings may be had by histologic study of sections. This is always desirable where macerative changes may confuse the diagnosis.

I opine that such assiduous study to all aborted tissues would eventually substantiate the dictum of A. W. Meyer, et al., to wit: "How many cases of hydatiform degeneration we can find in abortuses in tubal or hysterectomy specimens even, will depend very much upon the care with which the examination is made, for the condition undoubtedly is extremely common and not rare as heretofore supposed."

Preservation of the mole may be made in Keiserling's solution No. 3—modified, or in ten per cent formaldehyde solution.

NOTE My: identification of immature forms in cases 2 and 3 was kindly verified by Professor A. W. Meyer of Stanford University.

CASE 1.

Mrs. L. C., age 27, spanish, married 9 years.

Admission diagnosis: Retained secundines. Admitted for removal of retained secundines.

Patient went to see her doctor about middle of October, 1929. Her last menstrual period was June 16, 1929. She had begun to "flow" but had no cramps. Her doctor told her she was between 3 and 4 months pregnant. She had a "cold," cough, vomiting and chills. He prescribed "tonic." Flow continued until Dec. 25, 1929. On Nov. 15, she called to see her physician about it but he was out of town. She saw no other. The flow from middle of October to December 25 was sufficient to require a napkin. Dec. 24, she passed a piece of "fleshy" substance from the vagnia with some large blood clots. Dec. 25, passed a large piece of fleshy substance about three inches by one, blanched and foul cmelling. I examined it and macroscopically it resembled placenta retained in the uterus for a very long time (macerated). She passed two small cysts on the morning of Dec. 25, but did not tell me until after her operation.

Family history: Father dead, cause unknown. Mother died of septicemia. No brothers or sisters. Husband in good health. Three children, ages 7 and 5 years and 1 year, 10 months, all living and well.

Past health: Measles in infancy, severe attack of of influenza in 1918, made good recovery; no other illness. Menses began at 18, every 30 days, regular, painless, 3 to 4 days duration. Last period, June 18, 1929. Three normal pregnancies and deliveries; able to nurse her three babies. Does not know of any disease of uterus, tubes or ovaries.

Present condition: Fair, anemic from slow steady flow lasting from middle of October; some uterine contractions.

No active lesion of nose, throat or mouth; teeth in fair condition. Lung sounds clear throughout, no areas of dullness.

Heart: no valvular lesions; no enlargement of borders, rythm normal. Abdomen: No enlargement of liver or spleen; some tympanites. Skin shows some blanching, suggesting anemia.

Special examination: The uterus was large, boggy and extended a little over half to umbilicus. The cervix was dilated to about size of one inch and the examining finger could just reach the edge of a retained placenta-like mass. Patient removed to hospital.

Pre-operative findings: As patient was being prepared for the operating room and after her preliminary hypo of morphine, she was seized with a hard uterine contraction and expelled a large hydatidiform mole, followed by profuse hemorrhage. Estimated weight of mole about two pounds.

Ether anesthesia. Cervix patulous uterine cavity had a peculiar feel to right side just above internal os, "gummy," not like ordinary placenta; this forced the finger to left side of fnndus, giving sensation of a bicornate uterus, but when mass was broken up the uterine cavity had normal contours. All parts of mole that could be detached were loosened with finger and carefully removed with placental forceps; uterus swabbed with dry gauze; half strength tr. iodine and iodoform gauze pack, light. Aseptic ergot (hypo); gauze removed in 24 hours, no bleeding; uneventful recovery.

Patient advised to report for examination twice monthly; also possibility of hysterectomy explained.

Catheter specimen of urine. Dec. 25, 1929; Reaction neutral; sp. grav., 1.005; alb., 0; sug. 0; epithelial cells, a few; pus cells, occasional.

Blood count Dec. 26, 1929: Red cells, 4,190,000; white, 14,500; hem. 75 per cent; polys, 74; col. index .9. On Dec. 28, 1929: Red cells 3,580,000; hem., 75 per cent; col. index, 1.07.

On January 25, 1930, uterus was involuting normally and there was no evidence of chorion epithelioma.

CASE 2

Mrs. "A. G. X.," white female, age 34. Early history not relevant.

Menstruated first at eleven and a half years; irregular all her life but conform most closely to 24 day type. Married at 29.

First Pregnancy, Dec., 1926. Abortion. Last menstruation Dec. 26; began to flow profusely Feb. 2nd and continued until 6th, then dark brown discharge; severe cramps night of Feb. 24; opiate for relief; cramps returned with profuse hemorrhage. Feb. 25 dilated and curetted uterine depth 4 inches, cervix patulous, decidual tissue removed which seemed to be disintergrated and broken into small pieces; some "membrane" had been expelled before operation. This was "spontaneous" abortion, a child was greatly desired.

"membrane" had been expelled before operation. This was "spontaneous" 'abortion, a child was greatly desired.

April 26, 1927, I operated her for chronic appendicitis and aberrant piece of mammary gland in left axilla (cystic degeneration). At that time left ovary contained a cyst about size of a thimble which was evacuated. Right tube normal, right ovary sclerotic. Small subserous fibroid, size of marble, in fundus of uterus removed.

Second Pregnancy, June, 1927. Abortion. Became pregnant in June, 1927; normal up to August; after a slight cathartic (cascara) "perfect decidua" passed with very little uterine cramping. (Had been given bromides and opiate to control pain).

In fall of 1927 she had what I diagnosed as membranous dysmenorrhea, passed membrane-like masses with three periods; advised curettage; refused. Used tr. iodine and ten per cent silver nitrate endometrium.

Third Pregnancy, March, 1928. Normal child. Pregnancy, March, 1928; baby born Jan. 7, 1929, ten days over calculated time; ROA. Threatened eclampsia, 25 per cent alb.; headache, electric lights before eyes; granular casts and pus in catheterized specimen of urine. All of this having developed in last few days of pregnancy. B. P., 138-78. Forceps delivery, laceration of cervix and perineum.

Fourth Pregnancy. Mole. Last menstruation Dec. 13, 1929. Noticed some blood stain Feb. 14, 1930; Feb. 19th, passed "mole," no pain, only dull ache (uterine sedatives had been given to prevent abortion). Sac about size of English walnut, no fetus could be located within it; mole degeneration of chorion detected by method of study outlined in this report. Passed fleshy particles for about one week. Feb. 24th, dilated and curetted. Small piece of mole degenerated decidua removed. Seventeen days later passed other fleshy masses.

NOTE: Sept. 27, 1930. For five months following the February curettement she passed membranous masses like those noted in 1927. She has a severe endometritis with laceration of the cervix and perineum, for which operative correction has been advised. Because she is desirous of another living child, operation has been deferred. She is now three and a half months pregnant.

CASE 3

Mrs. L. A. W., white female, age 34, born in Indiana. Family history irrelevant.

Only diseases of childhood are mumps and measles; always had good health; only sickness associated with pregnancy and appendicitis, July, 1921. First menstruated at age of 15 years, regular, 28 day type. Married in 1918 at age of 22.

First Pregnancy, delivered Nov., 1919. normal. Child weighing ten and a half pounds—no trouble at confinement.

Second Pregnancy, Nov. 16, 1919. "Mole." Went to see Dr. Charles C. Wallin, Lewiston, Motana, in April, still nursing first baby. Had dizzy spells, felt badly whole time, nauseated, tired, dragged out. Two months later flowed for 3 weeks; stopped, no flow from April to Nov. Sudden severe hemorrhage. Dr. Wallin states, "Mrs. L. A. W. entered the hospital Nov. 15, 1920, with uterus enlarged due to five months pregnancy She had moderate continual vaginal bleeding and pain; no fetal heart sounds, no fetal motions and no ballottement. The uterus was heavy and boggy, about one finger dilatation of the cervix. The vagina was packed for 24 hours, at the end of which time, sufficient softening and dilatation had occurred to permit manual dilatation and removal of mole. Convalescence entirely affable."

Third Pregnancy. Abortion, Aug. 21, 1921. Report of Dr. C. C. Wallin: "July 22, 1921, she was re-admitted to the hospital with severe attacks of pain in the appendix region: white count, 17,450. This was the third and most severe attack she had had. She was at this time three months pregnant. I removed her appendix and she left the hospital August first. On Aug. 17, 1921, she returned to the hospital with slight bleeding and some lower abdominal pain, temperature 100. On the 21st her temperature went up to 103, pulse 120 and she had a chill. I cleaned her out the next morning and her temperature came down to normal. She left the hospital in good condition on the 25th of August. There were no palpable pelvic abnormalities at the time of the appendectomy."

Later, patient had pelvic reair work done in South Bend, Indiana.

Fourth Pregnancy. Mole; May 30, 1922. Report of Dr. Fred F. Attix, Lewiston, Montana: "We delivered a big hydatidiform mole from Mrs. L. A. W. May 30, 1922. As this was not a hospital case, our records are not complete."

Patient thought that she was about four months pregnant and that she had severe hemorrhage several days before mole was expelled. She further stated that Dr. Attix advised hysterectomy.

Fifth Pregnancy. Normal; May 26, 1924. Normal pregnancy, small child weighing four and a half pounds. Nothing abnormal.

Sixth Pregnnacy. Normal; March 26, 1925. Normal child born March 26, 1925, weight seven pounds. Nothing abnormal recorded about pregnancy.

Seventh Pregnancy. Normal; June 6, 1926. Normal child delivered June 6, 1926, weighing six and three-fourth pounds. Nothing abnormal recorded.

Eighth Pregnancy. Mole; April, 1927. Patient states she was about three months pregnant when she expelled the mole. She flowed for six weeks before she expelled mole,—"packed in ice" several days to check hemorrhage. Recovery prompt; mole exelled in one piece.

Ninth Pregnancy. Abortion; Sept., 1927. Three months pregnant; had uterine contractions, cause undetermined. Sac ruptured, expelling fetus; placenta retained, curettement in San Francisco, Cal. This was not a mole.

Tenth Pregnancy. Miscarriage; June, 1928. She was six months pregnant, when spontaneous rupture of membranes occurred and labor followed in three days. Macerated fetus. Infection followed, had fever for about one week.

Eleventh Pregnancy. Normal; June 14, 1929. Full term pregnancy, delivered of a healthy infant weighing five pounds. For the first three months of this pregnancy she flowed off and on every few days, just enough to soil a pad; there was one heavy flowing spell. Throughout, there was an unfortunate phobia due to her previous experiences with moles. Uterine inertia at the time of delivery resulted in protracted labor. Conditions of placenta not known.

Twelfth Pregnancy. Mole; March 16, 1930. While she was still nursing her baby born June 14, 1929, she menstruated in November, 1929. December 14, 1929, she had a "normal" menstruation but she was nauseated. January 18, 1930, she had an alarm-hemorrhage. Cervix and vagina packed with gauze and patient rushed from her ranch home, four miles out in the country, to hospital. Glucose and salt solution given to build up blood volume. In ether anesthesia, retained secundines, small immature mole removed; uterus swabbed out with gauze wipe, then swabbed out with gauze contining half strength tr. iodine; then packed with iodoform gauze for 24 hours. Two weeks previous to this she expelled small masses of tissue and immediate operation was advised but refused on the ground that she did the same thing in her eleventh pregnancy and was delivered of a normal child at term. Uneventful convalescence. All blood clots and tissue masses were examined carefully and "fleshy" parts washed —no fetus was found. Mole degeneration (immature) of chorion detected by method of study outlined in this report and verified by histologic sections.

CONCLUSIONS

1. The severity of the hemorrhage is no criterion of the size or age of the mole.

2. The typical large hydatidiform mole is an end product which has taken months to develop. (Meyer.)

3. The small, immature mole is none the less a mole, with all the syncytial potentialities of the mature mole. It may often be identified by the simple method of gross study herein described.

4. Careful inspection of all spontaneously aborted tissue will

(a) Increase the incidence of hydatidiform mole to "extremely common" (Meyer);

(b) Lower the mortality figures usually given;

(c) Substantiate the observations of Cierse (1847) that moles constitute a most common cause of abortion in the early months.

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DISCUSSION

DR. FLOYD B. SHARP, Phoenix: After hearing Dr. Yount's paper, I am much impressed with the amount of work he has done on a subject which is extremely complicated in its many aspects. The study of hydatidiform mole as a clinical entity, its incidence, cause, diagnosis, and the best treatment is very difficult when searching the current literature. The gleaning of conclusive statistics is almost impossible because of the extreme variation in the experience of observers. One is struck, however, by the recent renewed interest in the disease. Attempts are now being made to outline methods for accurate study, and I believe that Dr. Yount's plea for more careful study of tissue from abortions is an important contribution to our line of thought.

As to incidence of hydatidiform mole, it is almost certainly true that our lack of exact knowledge on this item is due to our universal failure to study thoroughly so common a thing as an abortion, and to our failure to report cases of mole without complication or unusual gravity. It is probable that the growth of interest in obstetrics and the stimulation of the cancer research movement will call the attention of practitioners to the importance of closer study of clinical material and the reporting of cases of this kind. It is certain that the incidence of mole is greater than formerly believed and the importance of the condition is emphasized by the demonstration that nearly fifty per cent of chorioepitheliomata are preceded by mole. Authorities believe that this could be raised to one hundred per cent if proper observation into the past medical histories could be made.

Etiology of the condition is likewise not fully known. In addition to conditions set forth in text books it has been interesting to note that in two of the three cases of advanced mole, which I have seen and treated during the last four years, various drugs to induce abortion had been taken early in pregnancy. The third case had had a general anesthetic a few days after her first missed period.

Early diagnosis of the condition is not easy except in rare cases of passage of portions of the tumor. As in all potentially malignant conditions this is the ideal time to discover and treat them. Special attention should be paid to irregular bleeding, rapid increase in size of the uterus, and marked interference with the general health. Realization of the nature of the condition in advanced cases is usually easier, but the treatment more difficult.

Proper treatment, whether by radical or more conservative methods is still in dispute. The good surgeon and obstetrician will adapt the method best suited to the individual case. Again, it is here that more definite knowledge as to sequelae, complications, and results of different methods could be of help.

I am heartily in accord with the thought of Dr. Yount, that there is much to be gained by closer and more systematic study of material which probably passes under our eyes more frequently than we now think.

DR. R. H. THIGPEN, Jerome: I had a case of hydatidiform mole I should like to report. A Mexican woman who had a large family by a former marriage was supposedly pregnant for twelve months with irregular bleeding throughout that time. She was much larger than she had been for her normal pregnancies. An enormous uterine tumor was found and I did a good deal of curettage which I know now was not good management. The woman had a normal pregnancy and delivery not long afterward. The surprising point in this case was the duration of the pregnancy and the patient's increase in size over her normal pregnancies.

DR. H. A. REESE, Yuma: A well developed Mexican woman, the mother of several children, came to me for care during a pregnancy. I suspected hydatidiform mole and treated her expectantly. Later she seemed to terminate her pregnancy satisfactorily as a miscarriage. I observed her carefully over a period of one or two years but no malignancy was evident. I should like to emphasize the danger of malignancy in these cases, and the importance of observation over an extended period of time. Diagnosis can be made early in cases in which two or three of these moles are passed. The physician is then on the lookout for hydatidiform mole, for which this symptom gives the clue. The condition is very rare. My own experience has shown just one case in the one thousand cases I have attended. I should like to see by a show of hands how many of the doctors present have seen one or more cases of this sort. (Hands of about half of the group were raised). I see that the condition is not so rare as I had supposed.

DR. JOHN E. BACON, Miami: Ten years ago a healthy young woman was brought to the mining camp as a bride. Six or eight months later a spontaneous abortion terminated a case of hydatidiform mole. I did not know to do as Dr. Yount has suggested. Ten months later tuberculosis was diagnosed at camp and she was sent to Dr. Pottenger's Sanatorium where a diagnosis of malignancy of the chest was made.

Another woman was given proper diagnosis because of her symptoms. She had a terrifying hemorrhage and a cesarean section was performed. She has had two or three healthy little Mormons since.

Fig. 1. Normal villi two months gestation (magnified nine diam.).

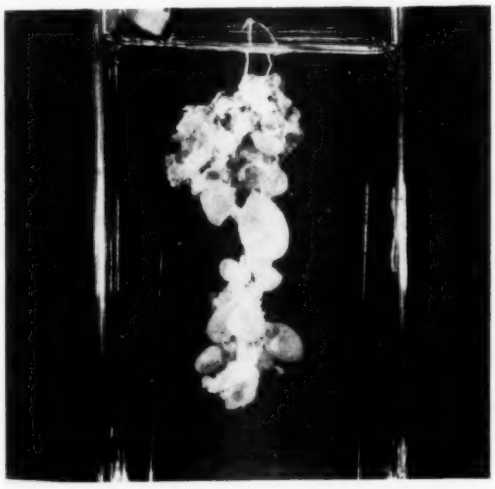

Fig. 2. (Case 1). Hydatidiform mole, full blown form. Shows about one square inch of mole matrix suspended in solution, representing approximately one-thirtieth of the whole mole. About five months gestation.

Fig. 3. (Case 2). Immature form of hydatidiform mole. Anatomical age about five weeks, showing hydatidiform degeneration and some slight changes due to maceration; also a few normal villi. (Magnified nine diam.).

Fig. 4. (Case 3). Immature form of hydatidiform mole. Menstrual age of mole two or possibly three months; twelfth pregnancy and fourth mole. The bladder-like degenerations are easly recognized with hand lens. (Magnified nine diam.).

THE SIGNIFICANCE OF HEMOPTYSIS

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(Read before the New Mexico Medical Society, at its Forty-eighth Annual Meeting, Raton, N. M., June 4, 5, 6, 1930.)

The spitting or coughing of blood in greater or lesser quantities is a frequently observed phenomenon, but there are few symptoms which may have a more varied origin. Various text books tell us that there are from twenty to forty different pathologic entities which may manifest themselves in this way as one of their symptoms. It is, therefore, a matter of importance that we should not come to any hasty conclusion in the presence of hemoptysis, whether scanty or profuse, temporary or constant, but, bearing in mind the many conditions with which it may be associated, endeavor by a process of elimination to differentiate the actual underlying cause.

The proper evaluation of blood spitting is not always an easy matter. Many physicians treat it lightly and tell the patient it is a little bleeding from the throat which will clear up. Blood-spitting, unless its presence can clearly and without any doubt be accounted for by some local specific condition, is never a light matter and it is a serious wrong to a patient to treat it with indifference and not endeavor to trace the condition from which it originates. Even one solitary hemoptysis in the history of a patient may have a very serious significance. It may betray the presence of an unsuspected neoplasm in the early stages. The point to be stressed is that bloody expectoration is never under any circumstances a matter that should be allowed to go unnoticed without a satisfactory reason for its occurrence being sought and established.

In this paper we shall attempt to point out the most prominent disease conditions which may have blood-spitting as a symptom.

HEMOPTYSIS AS A SYMPTOM

Probably nine-tenths of hemoptyses are associated with lung disease and especially with pulmonary tuberculosis. While blood-spitting is commonly associated with pulmonary tuberculosis it is injudicious to associate them as cause and effect without verification. Crofton[1] states that the records of the Veterans' Bureau show that while eighty per cent of patients examined for tuberculosis show blood-spitting only twenty-five per cent of them have tuberculosis. In actual practice it will be found that from thirty to eighty per cent of all cases of hemoptysis are cases of pulmonary tuberculosis. In the latter disease blood-spitting occurs sooner or later in from sixty to eighty per cent of such cases. It makes little difference whether the bleeding results from actual invasion of the lung tissues or from the rupture of a tuberculous bronchial gland into a bronchus.

Broncho-pulmonary hemorrhage, without definite symptoms or signs of cardiac disease or ulcerous pulmonary disease, is due, in the very great majority of cases, to tuberculosis; and, even if this cannot be demonstrated at the time, it may, however, be demonstrated later or be verified at autopsy, as has happened in many reported cases. In early tuberculosis the sputum may be streaked with slight, bright red blood while in advanced cases the blood is dark red and copious from erosion of small arteries by tubercles.

While the presumption is strong that any patient with hemoptysis (if there is other supportive evidence) is tuberculous yet we would like to refer to the other conditions that should be excluded.

Bartlett[2] points out that hemorrhage from the lungs may be divided into four classes:

Hemorrhage due to some constitutional or blood disease;

Hemorrhage due to disease of the heart or blood vessels;

Hemorrhage due to disease of the lungs;

Hemorrhage due to miscellaneous causes.

Any valvular disease that has progressed far enough to produce engorgement and congestion of the lungs may give rise to blood spitting. In acute congestion the blood is bright red and frothy. It need not, however, as Sir St. Clair Thompson[3] remarks, be necessarily frothy nor need it be coughed up. The blood may just well up. This writer points out that hemorrhage from the lungs may be profuse and yet neither auscultation, percussion nor x-rays may be able to detect any physical sign of its origin. Hemoptysis may appear in emphysema, influenza, pneumonia, acute and chronic bronchitis, bronchiectasis, infection of the lungs, syphilis of the lungs, localized pneumothorax. Lung abscess, lung gangrene, or neoplasms in the early stage, when their presence may not be revealable by x-ray, may be accompanied by hemoptysis. A brisk hemorrhage may be the initial symptom of lobar pneumonia. Asthmatic paroxysms may be accompanied by blood streaked sputum. In the case of neoplasm there may only be one or two isolated hemorrhages, and such a history is important.

Among the constitutional conditions which may give rise to hemoptysis, we may class

those persons known as "bleeders." Libman and Ottenberg[4] report the occurrence of hereditary profuse hemoptysis in seven members of a family in which tuberculosis was excluded. The cause of the hemoptysis was unknown. Cases of vicarious menstruation occurring as hemoptysis, such as that reported by Chapman[5], may also come under this connection. These hemoptyses occur only at the menstrual periods. In the writer's opinion any blood spitting occurring at this time is likely to come from a pulmonary tuberculous lesion, not necessarily demonstrable.

HEMOPTYSIS AND HEART DISEASE

A special word should be said in regard to hemoptysis associated with heart and vascular diseases. Repeated moderately large hemorrhages may at times be observed in young subjects, apparently coming from the lungs and giving rise to the suspicion of tuberculosis. Examination may, however, show the bronchial markings unusually increased and the size, and sounds of the heart altered. In these cases the causative lesion may be found to be an endocarditis, the sequel of some infectious disease, or a mitral stenosis. Rist and Rolland[7], in five cases of streptococcal endocarditis exhibiting hemoptysis, found infarcts in the lung in two. Duken's case[7] of recurring polyarthritis with endocarditis of infectious origin showed at autopsy a hemorrhagic infarct in the lower lobe of the right lung.

In Ramond's case[8], the patient, a youth of seventeen years, had suffered from scarlatina and chorea, the latter of which as the literature shows is a great producer of cardiac disturbance (choreic endocarditis). Examination showed mitral stenosis. The cardiac conditions were tolerated fairly well until the boy went to work two years ago when the exertion brought on severe hemoptysis. The heart could not stand the strain and pulmonary apoplexy was produced with infarct resulting in the hemoptysis.

In McMurry's[9] five cases of hemoptysis of circulatory origin three showed mitral stenosis, one aortic regurgitation with mitral insufficiency and one decompensation. In three of these cases a previous diagnosis of pulmonary tuberculosis had been made owing to the hemoptysis. The early morning rusty sputum is characteristic in this type of case.

Embolism and infarct in a pulmonary vessel, if not immediately fatal, may give rise to hemoptysis. Also the rupture of an aneurism, as in the case reported by Stewart[10] or leakage or pressure from an aneurism. Atheroma and high blood pressure can also be causative of hemoptysis.

A digression may be made here for a moment to consider the relative frequency with which hemorrhage is found in pulmonary tuberculosis, and other conditions in which the lungs are affected. Vinson[11] of the Mayo Clinic compared one hundred patients with pulmonary tuberculosis, one hundred with chronic bronchiectasis and one hundred with mitral stenosis with a view of observing the relative frequency of hemoptysis in these conditions. As far as could be observed the lesions mentioned were the only ones from which the selected patients suffered. Hemorrhage was observed in twenty-nine of the tuberculous patients, in eighteen of which it was moderate to severe. Pulmonary hemorrhage was found to be most frequent and most severe in the bronchiectatic patients; i. e. forty-nine cases, twenty-nine of which were moderate to severe. Only eighteen of the mitral stenosis patients showed a sputum streaked with blood.

HEMOPTYSIS FROM CONDITIONS IN UPPER RESPIRATORY TRACT

Distinction can usually be made between a hemoptysis originating from some condition in the throat and a pulmonary hemorrhage. The blood expelled from a non-pulmonary source cannot be aerated and is dark in color. This darkened sputum stops when the hemorrhage ceases, but in cases where a dark blood stained sputum originates from a pulmonary hemorrhage it continues for about twenty-four hours following the hemorrhage.

Sir St. Clair Thompson states that a good method of distinguishing whether blood comes from the lungs or bucco-pharynx is to throw the sputum into water. If the blood comes from the pharynx, larynx or gums, it is at once dissolved on shaking in the water; if it comes from the lungs it remains in one mass and insoluble. Profuse dark blood spitting seldom comes from affections of the upper respiratory tract. Bloody sputum may, of course, be associated with an epistaxis when the blood drains backward into the pharynx. The more usual causes of hemoptysis originating in the upper respiratory tract are associated with varices in the pharynx and base of the tongue, with hemorrhagic laryngitis, papilloma of trachea, neoplasms, foreign bodies, congestion from pressure of enlarged thyroid or aneurism.

HEMOPTYSIS DUE TO PARASITIC DISEASES

In a very extensive article Castellani[12], formerly professor of tropical diseases in the Tulane University of Louisiana, says: "It would seem that the subject of hemorrhagic bronchitis of protozoal, helminthic and fungal origin deserves to attract more attention than has been the case hitherto. It should be remembered that the subject is of prac-

tical importance as all these conditions which I have mentioned are generally mistaken for tuberculosis."

Castellani particularly describes bronchospirochetosis and broncho-moniliasis, accompanied by hemoptysis. While such conditions are more usually observed in tropical countries, the mycotic diseases are not unusual here. Bridge[13] reported seven cases of actinomycosis five of which showed hemoptysis. Jaureguy[14] reported eight cases in hydatid disease, this condition not infrequently occurring in the United States.

MISCELLANEOUS CAUSES OF HEMOTYPSIS

There are numerous other conditions which may be causative of hemoptysis. Among these are various blood conditions, such as purpura, scurvy, pernicious anemia, leukemia. Rist and Rolland[5] say that changes in the blood, such as anemia from destruction of red blood corpuscles, or azotemia, may possibly be able to provoke hemorrhage independently of either an infarct or endocarditis of right heart. Mercurial stomatitis, phosphorus poisoning, cirrhosis of kidney or liver, and certain acute fevers may show hemoptysis. In stomach and duodenal ulcer and typhoid fever blood is frequently passed by the mouth but in these cases the condition is a hematemesis rather than a hemoptysis. Gout, according to Semon[15], is a fertile source of pharyngeal hemorrhage. A minor unsuspected rib fracture may be the underlying cause.

DIAGNOSIS

Cases of persistent hemoptysis, in which pulmonary tuberculosis and heart disease can be definitely excluded, should be thoroughly investigated with the bronchoscope, bearing in mind the various conditions mentioned in which hemoptysis may occur. Special clinical diagnostic criteria for such special conditions should be tested. Chevalier Jackson[15] gives as an indication for bronchoscopy the appearance of any suspicious shadow on the roentgenogram. Endoscopy may be the only means of correct diagnosis. In some cases injection of the bronchial arborization with lipiodol has been advocated. The writers[17] have been opposed to this procedure in cases of hemoptysis but recommend waiting at least a month after cessation of blood spitting before the use of iodized oil as a diagnostic method. The presence of unsuspected foreign bodies may be indicated on a roentgenogram. Some conditions that give rise to hemoptysis, such as carcinoma of a bronchus, are not always demonstrable premortem.

TREATMENT

In the case of hemoptysis as a part of the syndrome of pulmonary tuberculosis the treatment, of course, is part of the generally recognized treatment of this disease.

In the case of non-tuberculous pulmonary disease the treatment will depend upon the degree of hemoptysis and the nature of the underlying cause. Astringent and coagulating drugs may be used to control the bleeding. Sir St. Clair Thompson recommends adrenalin as an astringent. He also says that in moderate hemorrhage a hypodermic injection of morphine, (gr 1-6 to 1-4) with atropin (gr. 1-200 to 1-150) is one of the readiest, quickest and most reliable remedies.

Among the newer drugs Houssay[19] and Pissavy[18] recommend hypophyseal medication; gelatinized serum is recommended by Pierre[20] and many others. The new calcium salt, (calcium gluconate) is highly favored as a rapid coagulant and may be injected intramuscularly without harm. The intravenous use of calcium chloride is an old remedy. In the experience of the writers drugs have proven of limited value, except morphia to quiet the patient. When the bleeding persists in spite of absolute bed rest with shoulders elevated so that expectoration may be accomplished with the least effort, surgical methods should be adopted at once such as pneumothorax or phrenicotomy.

Wiggers[21] of Ann Arbor, who made a very thorough physiologic investigation of the treatment of hemoptysis, says, "Early in the course of hemoptysis when the breathing has not altered except for an occasional cough, one is dealing practically with a normal subject and the object of paramount importance is to reduce the bleeding by drugs that lower the pressure within the pulmonary circulation. Investigation has shown that this cannot be accomplished by vasomotor drugs such as the nitrites and that cardiac depressants, such as chloroform and pituitary extract, must be resorted to. If hemorrhage has continued till the heart is very rapid and respiration is accelerated with anemia of the cerebral center, pituitary extract is the only remedy that will elevate systemic arterial pressure and at the same time lower it in the pulmonary circuit."

In hemoptysis due to heart conditions Ramond[3] recommends absolute rest in bed, careful dieting and digitalis as immediate and continued measures. He thinks that a pulmonary infarct (due to cardiac conditions) has a natural tendency to recover unless associated with asystolia.

In moderate hemoptysis, when an artificial pneumothorax is not possible, Knoll[22] recommends the application of compressing rubber bandages on the affected side so as to suppress completely the respiratory movements on this side and on the diaphragm.

Surgery has now a prominent and important place in the treatment of serious hemoptysis. If large vessels are eroded, tying the external or common carotid may be necessary. Pneumothorax and phrenicotomy or evulsion of the phrenic nerve have given excellent results, a fact well known to this audience. Tuffier[23] suggested and practiced pleurolysis in preference to artificial pneumothorax. Dalton[24] found venesection successful in a case of hemoptysis with mitral stenosis.

Beye[25] says that ligation of the pulmonary artery has not been successful in controlling cases of lung infection. In two cases reported, however, ligation of the branch of the pulmonary artery to the lower lobe of the right lung was attempted to control hemorrhage. In these cases a partial thoracoplasty and phrenic neurectomy had been tried and failed. The author considered the operation justifiable in such cases when the hemorrhage is coming from the branch of the pulmonary artery or pulmonary vein. If it is coming from a bronchial vessel ligation of the pulmonary artery will do no good. In his two cases Beye found this method successful.

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DISCUSSION

DR. R. O. BROWN, Santa Fe: Three members of my own family, including myself, started our residence in the Southwest by reason of a little blood in the sputum. I have a sister-in-law who had 17 full-sized hemorrhges before the doctor admitted she had tuberculosis. I had four or five myself and an elder brother raised pinhead drops of blood in his sputum and after about thirty sputum examinations we discovered he had some tubercle bacilli in his sputum. In the years since 1916, since I have been out here, the largest part of my work has been in tuberculosis and when I come upon history of blood spitting that has been ascribed by doctors to some other cause, I think that is a matter of great importance, as in some of these cases later on when a pulmonary hemorrhage occurs, they go to a doctor who makes a more thorough examination with diagnosis of tuberculosis, which is anywhere from a year to four or five years after it should have been made. It is rather difficult at times to recognize tuberculosis; sometimes it seems no job to me at all, while at others it is quite difficult and I rather suspect that there are times when it is impossible for us to find it, yet it still exists.

DR. F. D. VICKERS, Deming, N. M.: I think this a very important paper. Some of these cases are extremely interesting and it is very difficult to definitely diagnose them. I have a case now which raises a little blood from the right lung, which I am sure is bronchitis. I think we in the Southwest are prone to think that all these cases are tuberculous, when many times they are not.

DR. J. M. BRITTON, El Paso, Texas: We have many tuberculars in El Paso and every now and then some patient will come into my office who has had a slight hemorrhage which he thinks is from the nose or throat. He will say, "I have had a little hemorrhage which I think was from my nose." It is probably only one case in a thousand in which the homeorrage is from the nose or throat. If one could just watch these cases, they would see that they turn out to be tuberculosis.

DR. R. J. GROOM, Santa Rita, N. M. I watched a very interesting case, a woman, aged 24, with tuberculosis. At each menstrual period, she would start in about ten days before her menstruation with cramps, nausea and vomiting, losing as much weight during that time as she would put on between times. We felt that it was advisable to bring about menopause, which we did with x-ray. After that she became an arrested case. Possibly about two years after she was pronounced arrested, she returned to her old home for a visit and while there commenced to spit up blood, so she came immediately. There was nothing in the physical examination or x-ray to account for this blood spitting. However, 28 days after she repeated the process, and for a year and a half that continued at those intervals. During that period we could never at any time ascertain any activity or chest finding that would account for the blood, and finally it ceased.

DR. F. M. HELLER, Pueblo, Colo.: It is easy to think in these cases of blood-spitting that every case is one of tuberculosis and the frequency with which this symptom is present is significant. I think, however, that among such cases are those of mycotic infections. Oftentimes individuals born and raised in Colorado will come into the office spitting blood and frequently on examination I find mycotic infections. These cases are extremely interesting; they have a mycotic process which produces a thin

spitting of bloody substance; it is mucus which is blood-stained. It is surprising also how many people who come to Colorado for tuberculosis have a mitral stenosis, which is usually of the type of enlarged heart on the left side, with which they often get in the mornings a bloody sputum. Then there are a lot of children who develop a lung condition, a chronic infection of the lungs, non-tuberculous, and these cases will often go on with exacerbation and an acute condition will develop with spitting of blood of a bronchitis nature.

DR. A. J. STREIT, Amarillo, Texas: In Amarillo we see very few cases of tuberculosis, but every once in awhile we do find patients who come to our office with spitting of blood. I remember one or two cases with mild bleeding from the septum and from the turbinates, some from the tonsils, and one case where it was coming from the upper part of the larnyx.

VINCENT'S INFECTION

R. J. GROOM, M. A., M. D.
Santa Rita, New Mexico

(Read before the New Mexico Medical Society, at its Forty-Eighth Annual Meeting, at Raton, N. M., June 4, 5, 6, 1930.)

INTRODUCTION

Vincent's infection includes more than Vincent's angina, and is an inflammatory lesion due to infection by the Plaut-Vincent fusiform bacillus and spirillum. While it is usually a disease of the mouth, Vincent's organisms normally inhabit several other locations and can produce lesions in any of these areas. It is characterized, first by an inflammatory process, later by the formation of a pseudo-membrane, and in the still later stages by the production of a sharply defined ulceration with characteristic "punched-out" appearance. While in the majority of cases numerous other bacteria may be found in the smears taken from the lesion, there is always a preponderance of Vincent's fusiform bacilli and spirilla.

ORGANISMS

The organisms associated with Vincent's infection are morphologically unlike. One is a short bacillus of fusiform shape, Gram negative in staining; the other is a spirillum which in general resembles that found in relapsing fever and is Gram positive. These organisms are anaerobic and can be cultured on a variety of media by anaerobic methods. There is still considerable dispute as to whether they are separate organisms or different cycles of the same organism. Tunnicliff believes that the spirillar forms are simply stages in the life cycle of the fusiform bacillus. Zinsser holds that they are separate and distinct organisms.

HABITAT

It is interesting to note that these spirilla are probably the organisms first seen in scrapings of tartar from the teeth by Leuwenhoek in 1683. They are found in relatively large numbers in tartar from carious and ill-kept teeth, and in tonsils and adenoids in a large per cent of the cases. Miller and Epstein found spirilla present in seventy-seven per cent of 160 apparently normal mouths and fusiform bacilli present in ninety-two per cent. Their presence in other localities of the body is not as generally recognized. Gifford reports finding both bacilli and spirilla in the conjunctival sac and meibomian glands. Pilet found them present in fifty-one per cent of preputial secretions of normal males and sixty per cent of the smears made from the material about the clitoris. The principal habitats of fusiform bacilli in the human body are:

1. The crypts of the palatine tonsil;
2. Tartar deposits on the teeth;
3. The smegma in the preputial sac and from about the clitoris;
4. And possibly in the intestinal tract.

DIAGNOSIS

There has been some little argument, since these organisms are so frequently found in apparently normal tissue, as to whether or not they are the cause of Vincent's disease or whether they are purely invaders. They are infrequently found in clean and healthy mouths but are almost always found in an infectious condition of the teeth, gums, and tonsils, and in Vincent's disease. While other organisms may be present, the spirilla and fusiform bacilli are by far the predominating organisms; and, as the infection or disease clears up, these organisms disappear. Epstein has said that these organisms are "opportunists" and that, when they do cause infection, it is in locations where they are normally found or perhaps transplanted to the lesions produced, and that they set up disease by taking advantage of the lowered resistance caused by such other infection.

Whether or not they are secondary invaders, they very frequently predominate the pathological picture and so are of preeminent importance.

They may be found in numerous diseases where they have no part in etiology, such as scarlet fever, streptococcal sore throat, carcinoma of the mouth, syphilis and tuberculosis of the throat. This, of course, is explained by the fact that the mouth is the normal habitat of these organisms.

LESIONS PRODUCED BY VINCENT'S ORGANISMS

I. Oral Passages:

(a) Vincent's angina, which at times may become epidemic and is the lesion in which these organisms were first recognized.

(b) Noma. That they are the etiological factor in the production of this disease

has been definitely established by Weaver and Tunnicliff.

(c) Pharyngomycosis, a disease characterized by the presence of actinomyces-like granules in the tonsillar crypts and an exudate on the tonsil and pharynx.

(d) Pyorrhea Alveolaris. For a long time these organisms have been regarded as of secondary importance in this disease, but they are so constantly present, and can account for the putridity of the lesions and the amount of necrosis seen in this disease, that they undoubtedly play a most important part in its production.

(c) Ulcerative Gingivitis or Trench Mouth. This condition became epidemic during the world war, and that these organisms are the cause of this disease was well established at that time.

II. Lesions of the Respiratory Tract. Their relation to the production of pulmonary gangrene and fetid bronchitis has been discussed by numerous authors. Pilot and Davis have reported thirty-seven cases of pulmonary gangrene due to Vincent's infection. Pulmonary abscess may follow tonsillectomy in cases infected with Vincent's, or ether pneumonia, perforating lesions of the trachea or bronchi, foreign bodies in the air passages, or other pulmonary infections, and are due to the organisms of Vincent's angina being carried in from the mouth by inhalation of droplets laden with these organisms. Possibly, in a few instances, the organisms may enter the lung as emboli from some other focus. However, it is very seldom that the organisms can be demonstrated in the blood stream.

III. Cerebral Lesions. There are a few cases reported in the literature of metastatic putrid brain abscesses, complicating putrid bronchiectasis, in which fusiform bacilli and spirilla have been demonstrated in the brain lesions.

IV. Lesions of the Ear. One fatal case of mastoiditis due to Vincent's organisms has been reported by Ottoni, and numerous cases of infection of the middle ear and sinuses are to be found in the literature.

V. Lesions of the Bone. A few cases of osteomyelitis of the inferior maxilla due to Vincent's infection following the extraction of teeth have been reported.

VI. Lesions of Lymphoid Tissue. Enlargement of the cervical and mediastinal glands due to infection by these organisms is rather common.

VII. Lesions of the Blood. Kilduffe is of the opinion, as is also Cottrell, that Vincent's organisms may be the causative factor in infectious mononucleosis, and Cottrell has demonstrated these organisms in smears from six cases. However, in metastatic lesions, such as those of the lung or brain, the organisms have not been demonstrated in the blood.

VIII. Genito-urinary Lesions. Gangrenous balanitis is not uncommon in cases with a long phimotic foreskin in connection with uncleanliness.

IX. Miscellaneous Lesions. Gangrenous infections of the finger due to Vincent's organisms being planted by biting the fingernails have been reported. In two of my cases of cholecystitis, Vincent's organisms have been demonstrated in the bile obtained by transduodenal drainage. In both cases these organisms were the predominant bacteria found.

SYMPTOMS

The symptoms vary with the location of the lesion. Malaise and fever are common in the acute infection. The temperature may reach as high as 104 or 105, although usually low. The pulse is ordinarily rapid. There is loss of appetite, disturbance of digestion; and, if the lesion is in the tonsils or gums, there is, ordinarily, severe pain in swallowing. The ulcerated surface bleeds readily on pressure.

The involved area at first is inflamed and swollen; later, becomes covered with a white or grey, loose, thin coating; by the third or fourth day the pseudomembrane becomes thick, white to greyish brown, and gives off a peculiarly offensive odor. The lymph glands draining the area are slightly enlarged. While, as a rule, the lesions are acute, they may become chronic in such forms as gingivitis, balanitis, bronchiectisis, and pyorrhea alveolaris.

DIFFERENTIAL DIAGNOSIS

In considering lesions, especially acute ulcerative ones, in any of the localities where Vincent's organisms are normally found, Vincent's infection must be considered. The chief lesions to be differentiated are diphtheria, tuberculosis of the throat, syphilis of the throat, acute tonsillitis, septic sore throat; and it must be remembered that, since Vincent's infection flares up where some other lesion has reduced the local resistance, not infrequently Vincent's disease may be present and may play a most important part in the pathology along with one or another of these infections. I have seen one fatality due to Vincent's angina superimposed on a tuberculous throat, flaring up and proving rapidly fatal after a cauterization. And two or three instances, where both diphtheria and Vincent's infection have been demonstrated, which proved fatal in spite of large doses of diphtheria antitoxin, I am convinced that death was due to the Vincent's rather than the diphtheria. The differential

diagnosis in these lesions rests on the characteristic pseudo-membrane and ulceration with a predominance of Vincent's organisms in the smear, as well as on the results of the treatment.

TREATMENT

In reviewing the literature on the treatment of Vincent's angina, we find that here, as in most diseases where there is no definitely proved specific, numerous therapeutic agents are claimed by various authors to be practically specific, and that the number of drugs recommended in the treatment of this disease is almost too great to mention. However, three drugs at present seem to be more used than all others; sodium perborate, arsphenamines and bismuth; perhaps the majority are using arsphenamines. Other drugs used to a considerable extent are various anilin dyes, especially methylene blue or gentian violet, potassium dichromate, and copper sulphate.

Personally, the best results that I have obtained have been with sodium perborate. I have been unable, either with ten per cent arsphenamine and glycerine used locally or arsphenamine intravenously to obtain the satisfactory results reported by its adherents; and when it is necessary to resort to hypodermic medication, as in cases of gangrene or bronchiectisis, I prefer bismuth intramuscularly to the arsphenamines. While we do not hesitate to give arsphenamine in syphilis, we must recognize that it is not without danger. Within the last month, I have had two untoward complications from the administration of neoarsphenamine, one a pulmonary embolus followed by pneumonia which fortunately did not prove fatal; the other, an acute nephritis. Not infrequent deaths have been reported following the administration of the arsphenamines.

The treatment advocated by Bloodgood, and which has proved so satisfactory in his hands that he insists that it is a specific, is sodium perborate. He makes a thick paste of the salt with water, applies it locally to the infected ulcerated areas, allowing this to remain in the mouth for about five minutes once a day; in addition he has the patient gargle a thinner solution two or three times a day. Sodium perborate itself is very disagreeable and unpleasant to use, and I have found some of the preparations which are aromatic more satisfactory. In only the severe cases is daily office treatment by the physician necessary. The mistake we make is in not using the solution of sodium perborate frequently enough; and, instead of gargling and rinsing the mouth two or three times a day, the patient should be instructed to use it in the mouth at hourly intervals. Treatment should be continued for several days after all lesions have disappeared and smears from the site of infection show no organisms.

That sodium perborate is a specific for Vincent's angina, I doubt; but since the organism can grow only in anaeroeic conditions, an oxidizing agent is logical treatment for the disease. It both removes necrosed material beneath which the organisms flourish, and produces oxygen in the otherwise anaerobic area, and by so changing the media, makes it impossible for the organisms to continue. The curative results in the use of sodium perborate are due to the change in the media where the infection is present, rather than to bactericidal action of the drug. I do not believe any remedy has yet been developed which is specific to Vincent's infection in the sense that quinine is in malaria. However, it is results and not methods that count.

PROPHYLAXIS

Since Vincent's organisms practically always are found in unclean mouths, the greatest prophylactic measure is, of course, cleanliness of the mouth with eradication of pyorrhea, decayed teeth, and infected tonsils whose deep crypts filled with carious material are ideal locations for the propagation of Vincent's. Since the war the instances of Vincent's infection have been markedly on the increase. However, we have seen fewer cases in the community where I live within the last year than we did in the previous two years. I attribute this to the fact that the dentist in this community has been an enthusiastic advocate of the use of sodium perborate as a mouthwash and uses it following all extractions and work on teeth in his office and has educated a very considerable part of the population to use a sodium perborate preparation more or less routinely as a mouth wash.

Soda fountains should all be required to use paper cups and dishes which can be destroyed. The habit of the use of a common water bag for a whole gang of working men should be discouraged, as both of these have been prolific sources in the spread of this disease.

CASE REPORTS

In reviewing the literature, I can find only one case reported of Vincent's angina in a toothless mouth, and that was in the case of a child six months old who has as yet cut no teeth.

Case 1. Male, white, age 53. Past history negative. His upper teeth had been extracted nine years previously and his lowers five year ago. His general health had been good. The patient came into the office complaining of very painful, sore throat. When first seen, tonsils and pharnyx were red and inflamed, but no patches of ulceration were visible. Temperature 102°; pulse 110°. On the second day grey brownish patches were found on the tonsils

and uvula, and by third day the mucous membrane of the nasopharynx was covered with a similar membrane. A diagnosis of diphtheria was considered, but cultures from both nose and throat were negative, and the smears showed only Vincent's organisms. On intensive treatment with sodium perborate of both nose and throat, pain rapidly subsided; in the course of five or six days the ulceration had practically cleared, temperature dropped to normal, and the patient felt greatly improved. He was advised to continue the treatment, however, and not to return to work for at least another week. Ten days later, he again reported complaining of malaise, lose of eighteen pounds weight in two weeks and was expectorating foul smelling sputum streaked with blood, which on examination contained fusiform bacilli and spirilla. The patient was started on intramuscular injections of bismuth in addition to continuing the use of sodium perborate in mouth and throat. Coughing and expectorating have lessened and he is gaining back weight and strength gradually.

Case 2. Child, seven years old. when first seen had typical sordes on the gums, pseudo-membrane on tonsil, and "punched out" ulceration on tongue and bucal surfaces. The throat was so sore that the child absolutely refused nourshment and was fed for a few days by rectum. The lesions were treated locally with 10 per cent arsphenamine in glycerine and 13 grams of neoarsphenamine were given intravenously at five day intervals. Later, the child was fed by stomach tube as she was unable to retain nutrient enema. In spite of treatment, she became rapidly more toxic and died on the tenth day.

Case 3. Child, three years old; when first seen had extensive greyish membrane on both tonsils. There was a case of diphtheria in the same family, and, without waiting for a report from culture, diphtheria antitoxin was administered. Report on culture when obtained was positive; however. smears from the throat showed only Vincent's organisms. Following the administration of antitoxin, the toxic symptoms disappeared to a considerable extent although the membrane did not disappear as rapidly as would be expected of a case of diphtheria under treatment; after three or four days extensive ulceration of the tonsils was present. The child's condition rapidly grew worse with marked prostration and toxemia with very little improvement in condition, in spite of both antitoxin and local application of arsphenamine; in the fourteeth day he died.

Case 4. Male, age twenty-eight, having far advanced pulmonary tuberculosis with some pharyngeal involvement. He had been in the Veteran's Bureau Hospital for the past two years, and showed marked pyorrhea alveolaris. Because of the pain from swallowing, the arytenoids had been cauterized. Painful swelling of the nasal pharynx soon developed and a thick, greyish membrane bleeding easily on pressure followed. Pain became more marked, the patient lost weight rapidly: temperature rose to 104° daily with rapid pulse. Smears from pharnyx showed a predominance of Vincent's organisms. Patient returned to the Veteran's Hospital where the condition was attributed entirely to tuberculosis: within seven days after the onset of pharyngeal lesion, the patient died. How much of his condition was due to tuberculosis and how much to superimposed Vincent's angina is problematic. However, this seems to be too fulminating a case for tuberculosis alone.

SUMMARY

1. Vincent's infection is very prevalent and at times a serious and even fatal disease.

2. The organisms are normally found in the presence of uncleanliness in various other localities than the mouth and can become etiological factors or disease in these locations.

3. Other infections may so change the media or lower the local resistance that the already present Vincent's infection may flare up and become actively pathological.

4. The most important treatment is prophylactic.

5. Sodium perborate has proved more satisfactory in the hands of the author in the treatment of Vincent's infection than any other remedy and holds less possibility of serious complications than the arsenicals.

6. The cure and prevention of the disease is brought about by change of the media so that the organisms can not thrive in their normal habitats.

DISCUSSION

DR. M. K. WLYDER, Albuquerque, N. M.: I think we have all enjoyed Dr. Groom's paper. I was very much interested in the case he mentioned of the man who developed a tingling in his arms and legs. I recently had a case of the same kind, a boy 12 years old. who had typical Vincent's, the throat and tonsils bleeding. The swab showed Vincent's organisms and we failed to get any diphtheria by swab and culture. However, with sodium perborate. the throat cleared up, but a few weeks later he developed typical post-diphtheria neuritis. I never heard of neuritis developing in these cases except the one the doctor mentioned and the one I had. It sems to me perhaps the Vincent's organism is more prolific and overshadows the diphtheria condition, because there were patches in the throat and though I had both swabs and cultures made, they were negative for diphtheria.

DR. A. J. STREIT, Amarillo, Texas: Dr. Groom has given us a very timely talk on this clinical condition and is one that may occur in the real young as well as the older children. There is usually quite a lot of pain. The child says, "Mother! my mouth hurts." Usually there is quite a lot of swelling around the gums. The temperature goes up to 101 or 102. according to the amount of involvement. This sort of infection may not always show upon making microscopical examination, so it has been my practice always to consider anything very susnicious where there has been a sore mouth for as long as four or five days. You will find these children usually come more or less from non-hygienic strata: they are more of an illiterate type: the mother is not careful of the diet. If you watch for these infections, you will usually find more of them in this type, particularly among the Mexican children who use a common drinking cup and patronize soda fountains which are not kept clean and where the hygienic conditions are not good. We ought to try and teach more the value of cleanliness.

DR. J. M. BRITTON, El Paso, Texas: I enjoyed Dr. Groom's paper very much. I have encountered many cases of this infection in my practice and my treatment is ten per cent copper sulphate. This acts like magic and then I follow it with hot gargle of sodium perborate which in nearly every instance affords immediate relief. For ulcers there is nothing like copper sulphate, 10 per cent. It clears them up right away. Do not be afraid to

smear it on a piece of cotton on a steel applicator and put it right down on the ulcers; then follow it with hot gargles of sodium perborate and you will get results.

DR. J. R. LEMMON, Amarillo, Texas: In a recent discussion at the New York Academy of Medicine, one series of cases was treated with salt solution, another series treated with sodium perborate, and still another series with arsphenamin. Those treated with normal salt solution got well just as soon as the other cases. We treat them first with tincture of iodine, then follow with Fowler's solution and then for home treatment ask the patient to use sodium perborate and usually have good results.

DR. F. D. VICKERS Deming, N. M.: I have a room in my office that is used by a dentist, who sees many cases of trench mouth. He uses perborate of soda, makes a paste of it and rubs it in, and usually they clear up very quickly under this treatment.

DR. GROOM (closing): There is still a question in my mind as to whether the case I mentioned was post-diphtheritic neuritis, or Vincent's, acting the same way. I am unable to find any cases in the literature to support our cases.

PREVENTIVE MEDICINE AS APPLIED TO PEDIATRICS

MELDRUM K. WYLDER, M. D.,
Albuquerque, N. M.

(Read before the New Mexico Medical Society, at its Forty-eighth Annual Meeting, Raton, N. M., June 4, 5, 6, 1930.

In these days we hear much of prophylaxis, health examinations and other preventive measures. Prevention of disease and suffering is the primary function of our Health Departments. Take prevention out of their work and they could not exist. Yet, with all the propaganda of prevention that has come on in recent years, there are many who think only of prevention in medicine as applied to birth control.

Pediatricians take and always have taken, the lead in preventive medicine. Pre-natal care is in the field of the obstetrician. By the proper supervision of the mother's diet, much can be accomplished and rickets can be prevented. By giving medicine to expectant mothers, cretinism, myxedema and muscular distrophies may be avoided. In known cases of syphilis in the parents, early and vigorous anti-luetic treatment will give us a baby free from syphilis. Birth injuries should be avoided if it is possible to do so, for they leave in their wake much suffering and anguish. I need hardly speak of the importance of cleanliness and asepsis in the avoidance of impetigo contagiosa, pemphigus, wound sepsis, erysipelas, conjunctivitis and stomatitis.

That splendid observer of the 18th century, Edward Jenner, brought out a prophylactic measure that has saved hundreds of millions of lives and untold suffering and by the universal use of vaccination against smallpox, this disease could be banished from the earth. This has been absolutely proven, and yet there are many otherwise intelligent people who fight vaccination. An hour could be spent on this subject alone. Diphtheria is another disease that can be banished from any community that will make the effort. The universal use of T.A.T. or Toxoid will accomplish this. We should bow our heads in shame and admit our failure whenever diphtheria is allowed to show its head, for T.A.T. should be given to every baby when six months of age; and six months is the time of choice, for a natural immunity exists up to that time. The earlier it is given the less reaction. In older children and adults the reaction will be more severe. An asthmatic or eczematous baby should be tested before giving the first injection. This can be done by first injecting 1/20 c.c., waiting one-half hour, then giving 1/10 c.c. and wait another hour and if there is no reaction, give the first injection. A week later give the second and in another week give the third injection. Four to six months later do a Schick test to see if the baby has acquired an immunity and if not, give a second course which will be almost universally successful. Always remember that the Schick material must be freshly prepared to be dependable.

The use of convalescent serum is now recognized as dependable. In poliomyelitis, if given intraspinally and early it is most valuable. The trouble is the difficulty of obtaining it. The large baby hospitals get all they can from their convalescent patients and keep it on hand but for the man in general practice it is hardly available. For measles it is available as there is always some child who has recently recovered from measles who can give a little blood. Two to five c.c. of serum from a patient who has recently recovered from measles will prevent it in another child in ninety-five per cent of cases, and where not entirely prevented, it will be so modified that it will not be serious. Think what this means in the case of a frail, under-nourished, tuberculous or athreptic baby. It means the difference between life and death. If a suitable donor who has had measles cannot be found, 30 c.c. of whole blood from a parent who has had measles, injected into the gluteal muscles has the same effect. Convalescent serum also works in mumps, chickenpox and encephalitis lethargica.

By giving iodine one week twice a year, we may keep up the iodine content of the thyroid and this will prevent many of the goiters of early life. Of pertussis vaccine we are not so sure, but it is the best thing we have

and in some cases it seems to give beautiful results; in others it fails. It should be used both as a preventive and curative agent.

No sane person now questions the efficiency of typhoid vaccine. Any community that will make the effort can now be free from this fever. In all the wars previous to the World War, typhoid fever killed more soldiers than the enemies' bullets. In the World War typhoid fever was ruled out. This is one of the contributions that Medicine gave toward winning the war. What other profession has done as much?

Tetanus antitoxin is a sure preventive, but the reaction is some times quite severe. As to scarlet fever serum, the best advice is that it should not be used as a preventive for the reaction is often more severe than a mild case of scarlet fever and its protection is for such a short time that the protection it gives is not worth the risk. It is, however, a big help as a curative agent in severe cases.

In hydrophobia the treatment must be instituted at once, especially if the bites are on any part of the body not covered by clothing, as these bites are always much more dangerous. The teeth of the biting animal in passing through the clothing leave much of the virus on the clothes. In order to be safe, when the bites are on the hands or face, begin treatment at once for it can be discontinued if the dog is proven to be free from rabies.

In tuberculosis the separation of infants from exposure to open cases is the best measure, together with sun baths. The feeding of tubercle bacilli of low virulence as advocated by Calmette and Guerin has not yet passed the Board of Censors and is not considered a safe procedure.

Rickets does not interest us much out here. Just let the baby get our sunshine and there will be few cases. However, in locations not so fortunate as we are, by the use of cod liver oil and viosterol, together with the little sunshine they have and the ultra violet light, rickets is preventable. And we, here, should give cod liver oil not so much for its vitamine D as our sunshine will prevent rickets, but for its vitamine A that raises immunity.

We give tomato juice and orange juice so commonly that scurvy is so rare few doctors would recognize it if they saw it. By clearing up the foci of infection we will reduce the number of cases of rheumatism and perhaps of chorea. However, the question of chorea is debatable.

Coeliac disease is probably preventable. These children are unable to digest starches. The tendency among some people during the second year to make up the diet principally, and sometimes entirely, of cereals, undoubtedly causes some of these cases.

Nervous disorders are a hard problem Nervous, irritable mothers raise nervous, irritable children, while on the other hand, parents that are cool, calm and deliberate in their actions, raise children of that same kind. This is part heredity and in part environment but you have all seen a fussy, nervous youngster taken to a hospital, or taken by an aunt who understood children and in a few days have the child's behaviour so changed you would not recognize it as the same individual. The prevention of behaviouristic and nervous troubles then lies in teaching the parents how to live with their children.

Marasmus or athrepsia is a conditiion we do not see as much as we used to since the balancing of the child's diet is becoming generally understood. By meeting the child's caloric needs, feeding the proper proportion of fats, sugar, protein and mineral salts and supplying the necessary vitamines and remembering that when a chidl does not thrive when given a proper diet there is always a reason. Look for this reason, clear up the foci of infection—it may be an ear, or a mastoid, or one of the accessory sinuses—and this condition will clear up. Keep the foci cleared up and these conditions will not develop.

Nutritional anemia results from keeping the child too long on a milk diet. By the early introduction of iron containing foods, this condition can be avoided.

Eczema can be prevented in those babies who show a tendency to exudative diathesis by feeding skimmed milk, as butter fat is often the cause of the irritatioin and, by increasing the amount of cod liver oil. This helps in two ways, first supplying a fat that is easier handled and increasing the vitamine A.

By boiling the milk we prevent septic sore throat, undulant fever, bovine tuberculosis and all milk borne infections.

I wonder if we are as interested in prevention as we are in curing. The prevention of disease is not as spectacular as its cure, but it is much surer and better for the patient and we are slowly but surely gaining ground. We are seeing diseases that were once dreaded, put in the class of preventable. Only by studying statistics can we get the true value of prevention and the physician who keeps his little charges out of danger is doing them a much greater service than the one who tries, too often in vain, to rescue them after the trouble has arisen.

DISCUSSION

DR. F. M. HELLER, Pueblo, Colo.: I think a paper from an obstetrician on the subject of preventive medicine is extremely interesting because of the fact that our advance in longevity has come so much through his efforts. We have increased the length of life because of the prenatal care in saving the child. I enjoyed the paper very much and only have a little to add, not to criticize. Dr. Wylder spoke of the problem of chorea and rheumatism in the child. That subject is open to a complete discussion insofar as we can expecct the mortality rate from heart disease, which is largely initiated in childhood, to be reduced. There is the infectious attacks on the valves of the heart, acute rheumatic fever. It has been attacked by the genera practitioner, by the pediatrician, and by the specialist, removal of the tonsils and no doubt every one has read in recent literature, upon the frequency of cardiac lesions in childhood after the tonsils have been removed.

Dr. Wylder did not speak on the one subject which to my mind is most important under the treatment of acute rheumatic fever, though of course this is still in doubt. To my mind when a child develops a fever, or a cold, or infection in the sinus of the nose and throat, it is most important that that child be kept in bed until the fever is absent. One thing the doctor mentioned only slightly, which to my mind is of great importance and that is frequency with which one will have a mastoiditis, often without pain or swelling, in which the symptoms are of the intestinal type. No doubt Dr. Wylder will be able to give us something on that subject, which will be of great interest.

DR. F. D. VICKERS, Deming, N. M.: When Dr. Wylder closes the discussion I hope he will tell us what kind of tonsils ought to be taken out, whether certain or all of them ought to be taken out, and if taken should stubs be taken out also, and if this is done, will it prevent rheumatic fever?

DR. J. M. BRITTON, El Paso, Texas: I want to say a word in regard to chorea. I have had a number of cases, one in particular a child 9 years old, the niece of my office girl, who had chorea for two years, the worst case I ever saw. She had very badly hypertrophied tonsils, badly infected, but her people hesitated about having them removed. I told them I could not promise them anything, but I felt that the tonsils should be removed. I operated that child and she was relieved immediately and has had no trouble since, though that was five years ago.

In regard to "blind" mastoids, we have one or two good men in El Paso, who are very good at that. I remember several cases last summer which were x-rayed and found immediately.

DR. C. F. MILLIGAN, Clayton, N. M.: I was very much interested in Dr. Wylder's paper. One phase I think we should stress and that is the mild cases. The severe cases of heart trouble and tuberculosis the doctor has mentioned will be brought to the specialists. The parents will realize that something is wrong with them and bring them in, but the general practitioner sees a few cases that are let go. The general practitioner has not time to bother with these things, but these cases will go on often into serious conditions if not taken care of. I think we should instruct our general practitioners to look after these cases more. We will do better work in preventive medicine if we take better care of these cases. Very few of our children are seen by the obstetrician; they are usually seen by the general man.

DR. A. B. STEWART, Raton, N. M.: Relative to the effect of heart disease in childhood, the greatest authority apparently on this subject today is Dr. Still of London, and his method of treatment is relatively simple. In childhood the use of salicylates is imperative, the same as mercury in syphilis. He has recently completed a new book which is most interesting and instructive on this subject.

DR. J. R. LEMMON, Amarillo, Texas: I have certainly enjoyed Dr. Wylder's paper very much and came all the way from Amarillo to hear it. I am ready to put my shoulder behind the wheel and help with this business of preventive medicine. I think doctors as a whole, especially general practitioners, are not enough sold on the idea of preventive medicine, and the sooner we are sold on this important part of medicine, the sooner we are going to be able to attain results and satisfy the laity.

In regard to the Dick theory, I have been an enthusiastic user of toxin and antitoxin for sometime. I have a little place on my history charts and always try to sell this to the people along with whooping cough vaccines. My experience with that has been that it will immunize people in 40 per cent of cases; in therapeutic measures I think it will 100 per cent. I have a hard job usually in getting patients to come back after a Schick test. I keep a list on my desk and have to keep checking them up. Most of them are very dilatory about coming in. The question of tonsillectomy in cases of chorea is almost universal. I believe unquesionably tonsillectomy will aid the average choreic child and that saline is still the preparation of choice.

DR. WYLDER (closing): I want to thank you for giving my paper such generous discussion. I am glad the question of "blind mastoid" was brought up. That is something we could spend an afternoon on. In any unexplained diarrhea cases, look at the mastoid and you may find the cause. If a child comes in with an unexplained diarrhea, if you will puncture the ear drum the diarrhea will clear up. Dr. Britton answered Dr. Vickers' questions in regard to tonsils and he can tell a great deal more about tonsils than I can. Still's works are certainly beautiful; he is a most interesting writer and I love to read all his books, but he does not advocate tonsillectomy as much as we do in this country. He has said the child should be sent to the seashore and on ocean trips before you take out the tonsils.

We are doing more and more in preventive medicine with the periodical physical examination and now we advise mothers to bring the babies in every month in order to supervise their diet and look them over and if we could only educate the American public to these things a great many upsets could be avoided.

I use toxin antitoxin because it came out first and the results are satisfactory. In regard to getting them to come in for the Schick test, I have had the same experience as Dr. Lemmon. My office assistant keeps a record and if they do not come in, writes them a letter to see if the child has developed immunity or not, and a great many of them will respond. However, a great many mothers will not have their children immunized for diphtheria; they do not have them vaccinated for small pox until they find they have to have a certificate for them to attend school. I think as near as I can tell about 15 per cent of the mothers I talk to about the advantages of diphtheria immunization bring their babies in for it. I believe, however, as the public becomes more and more educated, we will have better results.

SOME UROLOGICAL PROBLEMS OF INTEREST TO THE GENERAL PRACTITIONER

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(Read before the New Mexico Medical Society, at its Forty-eighth Annual Meeting, Raton, N. M., June 4, 5, 6, 1930.)

Diseases of the urinary tract are of interest to all physicians because of their direct and remote connection with the problems involved in all specialties. The general practitioner, however, shoulders the greatest responsibility, for it is his services which are sought by the patient at the onset of symptoms when the pathological lesion is in its incipient or early stage of development. In no other branch of medicine is the immediate recognition of symptoms, the accurate determination of their causes, and an early therapeutic attack of greater importance. It is encumbant upon all of us to be alert on all subjects, and profitable for all of us to consider frequently the meaning and significance of the outstanding urological symptoms.

HEMATURIA

I am prompted to discuss the subject of hematuria first, because it is a symptom which is, in about eighty per cent of cases, due to a serious pahtological condition. Yet our records show that the average case does not receive a thorough study until this symptom has existed on an average of over six months, the actual time varying from one day to twenty years. This is all the more regrettable, particularly in cases of tumor, because at the manifestation of first symptoms, the disease has already progressed to an advanced stage.

The list of diseases to be thought of and considered when blood is found in the urine is long and varied. It includes lesions of both the upper and lower urinary tract as well as certain constitutional diseases and conditions entirely remote from the kidney. The lesions involving the upper urinary tract include tumors, calculi, tuberculosis, nephritis, pyelonephritis, hydronephrosis, ureteral stricture and bilharzia. In hematuria originating from the lower urinary tract the causes may be neoplasm, polypi, paoilloma, caruncles, cancer, calculi, acute and chronic infections, ulcers, diverticuli, prostatic adenomas and carcinomas, seminal vesiculitis, and lesions in the urethra, such as infections, polypi, etc. Hematuria is also found secondary to systemic disturbances, such as hemophilia, purpura, scurvy, leukemia, infections, as appendicitis, secondary to the administration of insulin, toxins from dyes, etc.

This is an incomplete list of the diseases causing hematuria. While there are many causes, and it is necessary to think of them all, yet it is important to know that only three out of the many,—namely tumors, stone and tuberculosis are the existing causes in eighty per cent of all cases. Authors differ in the relative place in terms of frequency occupied by these three diseases. Tumors happen to take first place as the most frequent cause in my experience. It is to be remembered, however, that in eight out of ten cases consulting us for blood in the urine, the cause is a serious one and immediate action is necessary for its cure. It is disheartening to encounter cases particularly with inoperable malignant growths who give a definite history of hematuria from six months to a year with no effort having been made to determine the cause of the bleeding. On the contrary, it is a pleasure to encounter these cases at the onset of symptoms, find the small tumor, remove it by any one of the approved methods and enjoy the sensation of having saved a life from terrible suffering and an untimely death. Hematuria is the one urological symptom which should never be disregarded. Neither the patient nor the physician should be content until a positive explanatioin of its cause is known. The cause should never be guessed at. It is almost criminal to defer investigation, call it a strain, prescribe hexamethylenamine and await developments. The batting average of its being due to a serious cause in eighty per cent of cases is too great an odd against us.

I will not attempt to discuss fully the technique of the procedure of study in these cases. It includes macroscopic, microscopic and bacteriological examination of urine voided in the three glass test, a thorough physical examination, cystoscopic examination with pyelograms, comparative function tests, etc. While in most cases the location and cause of bleeding can be easily and positively determined, in others prolonged and detailed study is necessary. The very best time to make such a study is when the bleeding is active. There is great danger of error even after a thorough urological examination has been made. This is perhaps due to an inclination to pass judgment on a fogged or poor x-ray plate or rely upon one segregative function test or one bacteriological examination when experience proves that it frequently requires many efforts before the exciting cause is definitely found. Perhaps the tendency to do this is greater in urology than in some other branches because of the discomfort caused the patient

in obtaining specimens or making the pyelograms.

PAIN

Let us next consider the subject of pain, its causes and symptoms. Pain as a symptom in diseases of the urinary tract is, in many cases, very misleading and because of this responsible for many errors. It may vary from an intense colicky pain to a simple soreness or be entirely absent (even in serious lesions). When it is severe it is demanding and impels the patient to seek immediate medical aid. Unfortunately the lesions which cause the severest pain are the least imperative so far as the need for radical removal is concerned. The passage of a calculus causes perhaps the greatest pain yet such a case seldom demands an emergency operation. The more serious lesions, as tuberculosis, neoplasm, etc., may develop insidiously for months to years with an entire absence of pain. In fact, the first intimation the patient may have of the existence of such a lesion is hematuria, and the first pain caused by the passage of a blood clot down the ureter. This is most unfortunate because both of these diseases demand early radical removal if the patient's health and life are to be conserved. A small and innocent hydronephrosis may be accompanied by severe colicky pain which drives the patient to seek immediate assistance while a large hydronephrosis, so large as to cause pressure atrophy of the secreting kidney cells may exist for months with no pain. Not only does the type and severity of the pain vary greatly but its location is more apt to be remote from the urological organs than it is to be specifically associated with them. The ache in the lumbar region usually interpreted as a sign of kidney trouble has led to many diagnostic errors and is responsible for much self administered medication. On the other hand the absence of any such pain has allayed anxiety concerning the kidneys when in reality they were badly diseased and treatment was urgently and definitely indicated. It has been my experience that major kidney diseases are accompanied by pain referred to remote parts and simulate diseases of other organs, particularly the abdominal viscera, more often than to the so-called kidney area in the lumbar region. Conversely, diseases in other organs do cause pain which is referred to areas ordinarily interpreted as indicating lesions of the urinary organs. Pain in the lumbar region is more often caused by osteoarthritis than by lesions of the kidney and similarly some cases of pain in the perineum and urethra are caused by lesions of the sacral bones.

Lesions of the kidney may exist with negative urinary findings and yet be responsible for pain in remote areas as a hypernephroma with metastases in the skeletal bones. Because of these facts we cannot rely upon or place too much confidence upon any certain pain, or the absence of pain, when considering the urinary organs while making the invoice of our patient's disability. It is comforting to know, however, that it can be proven whether or not any given pain is of urological origin. The distention of the renal pelvis and ureter with pyelographic media in making routine examinations will, as a rule, reproduce the patient's pain and thus implicate the kidney as a source of that pain or serve as a strong evidence absolving the kidney in case the pain is not reproduced. In view of these facts it should be our rule to study the urinary organs in the presence of any pain, the cause of which cannot otherwise be positively proven.

DYSFUNCTIONS OF URINATION

Frequency, urgency, dribbling and all other phases of unnatural function of voiding, are more or less definite and indicate some involvement of the lower urinary tract. There is very little danger of being misled by these symptoms because their indications are direct and specific. It is to be remembered, however, that we may have very serious lesions involving the bladder, or prostate gland without any symptoms of dysfunction of urination. The most frequent of such conditions are tumors in the bladder, such as papilloma and even carcinoma. The so-called quiet prostatic hypertrophy with marked bladder distention and even carcinoma of the prostate. The routine examination of the urine showing blood, pus, or albumen, should furnish a clue which leads to the discovery of these conditions, for all such pathological elements found in the routine urine examinations indicate the need of a thorough cystoscopic study. It is to be remembered that the absence of these elements found in any single examination of a specimen of urine is not to be taken as evidence absolving the urinary organs of all pathological lesions. Frequently a complete negative urinary report may be rendered by the laboratory in cases of tumors of the kidney, or bladder, carcinoma of the prostate and even in calculi. It is not a rare experience to encounter such conditions of negative urinary findings with an absence of specific symptoms the patient reporting for examination because of general debility and the presence of these lesions only discovered by routine examination and these often repeated when the case was not satisfactor-

ily explained by the first effort. Experience has taught us the value of repeating the routine examinations of urine at regular frequent intervals—for pus, blood and albumen may show in some specimens and be absent from others.

PROSTATIC PROBLEMS

One of the outstanding urological problems with which we are confronted daily is that of the prostate gland. There are a few phases of this problem which I desire to emphasize. Its prevalence is not fully appreciated. Twelve per cent of all men past middle life develop some phase of prostatic obstruction. Many more men of all ages suffer from the direct and remote effects of follicular prostatic infections. We will discuss only the obstructing lesions. It has been a common practice to accept prostatic hypertrophy as an element of fate. Patients have been told that such trouble sare coincident to old age and something which they must accept and patiently endure. They have been taught to bear the discomfort caused by the obstruction as long as they can before resorting to radical measures for relief. I believe this to be a bad policy. The gradually increasing nocturia, starting with once a night and increasing to five or more times, is very disturbing to restful and restoring sleep. The back pressure of the progressively increasing amount of residual urine seriously interferes with kidney function and urinary retention products increase in the blood. These are toxic. They produce all the signs and conditions of senility, chief of which is increased blood pressure with renal, arterial and myocardial degeneration. Infections are bound to develop in the stagnant urine, stones are apt to form, the bladder wall becomse thickened and trabeculated, and diverticuli form and gradually enlarge. The longer the obstruction exists, the more pronounced these complications become so that by the time the symptoms are unendurable not only the urinary organs but other vital organs, especially those of the circulatory system, have become permanently and irreparably damaged. Such an advanced pathological state greatly increases the mortality rate and even worse, makes impossible normal functioning organs even in those who survive the extra hazard of the operation. This picture of delayed action in cases of prostatic enlargement becomes all the blacker when we consider that eventually twenty per cent of them become malignant. The increasing number of these cancer cases applying to the urological clinics today is appalling. While most of these are malignant from the start there are others which develop in the advanced adenomas. Not only all of this later type, but all of the above mentioned complications could be prevented if the obstructing prostate were removed early.

Consider the advantages of early removal. The patient is usually a good surgical risk, the operation can be done in one stage, the average hospital confinement is eighteen days, there are rarely any complications, and the patient is rewarded with normally functioning results. Contrast this with delayed action. The greater hazard, with the higher surgical mortality rate, a two stage operation with a much longer period of hospitalization, the greater tendency to serious post-operative complicatioins and the unsatisfactory functional results following.

In a consideration of these facts I am sure justice will demand that we advise our patients to attack with radical measure their prostatic problems at an early age. It is better to do this when they can than to wait until they are forced to.

I believe that urologists are making a great contribution to race betterment by conserving the health and vigor and by extending the days of active service of many old men whose advanced age has enabled them to accumulate knowledge and develop wisdom. Every patient complaining of nocturia, with hesitancy, diminished stream, or urgency, should have a careful examination. All such cases are not due to true hypertrophy. Many of them are simply congested glands which, if not relieved, become infected and definite hyperplasia develops. Simple bar obstruction is responsible for these symptoms in many cases. This condition is now simply, safely, and efficiently removed by the cautery punch technique. By this method, applied early, many more serious operations, which later become necessary, may be prevented.

The cancer phase of this prostatic problem is worthy of more detailed discussion. I can only invite your attention to the large incidence of this condition and urge that it be thought of in connection with the physical invoice of every man past forty-five. Let me explain that many of these cases are well advanced before any clinical symptom develops. It is not unusual to encounter patients whose only symptoms are caused by metastatic growths in the bones and who have no symptoms of dysfunction of urination and no abnormal findings in the urine. This is the treacherous phase of this problem. Canver of the prostate may insidiously develop into an advanced stage without producing any local symptoms. In the majority of cases it can be detected by rectal palpation. It is

characterized by a flatness and definite hardness. It is more or less fixed and has an indefinite border outline and at times distinct, localized indurated areas. There are cases in which the malignancy develops in the center of an adenomatous gland. These cannot be detected in their early stage by any method of examination which is a big argument in favor of the early removal of all adenomatous glands. While malignancy of the prostate may exist even with the entire absence of any disturbing symptoms, in contrast to this we may have marked disturbance, as nocturia, dysuria, frequency, urgency, pain, priapism, etc., when the prostate may feel very small, no marked induration detectable, and there is practically no residual urine, the symptoms being all out of proportion to our examination findings. Here, again, the final answer is to be had only by repeated observations and study. Palpation of the prostate through the rectum ought to be made a part of every general examination.

TUBERCULOSIS

We will not attempt an extensive discussion of urinary tuberculosis. The big problem with this, as in all of the others to be emphasized, is an early diagnosis. Fortunately renal tuberculosis is usually unilateral at first. Tuberculous lesions of the kidney may develop very insidiously and continue until the kidney is extensively involved before any outstanding symptoms become manifest. We must bear this disease in mind in every case presenting symptoms of frequency, urgency and burning, particularly when such symptoms are accompanied by a few pus cells in the urine that is sterile to culture. After hematuria and the aggravating bladder symptoms develop the lesion is apt to have become very extensive, involving the ureter, bladder and sometimes the other kidney, making the securing of perfect therapeutic end results impossible. One must remember the great difficulty in finding the Koch bacillus in the urine as well as to demonstrate a lesion in all early cases by any and all of the urological methods. Negative findings in a suspicious case should never quiet our suspicions. Repeated efforts will reward us, by ultimately proving our contentions if the lesion actually exists. It is easy to establish a diagnosis with urological methods in advanced cases by pyelography, comparative function tests and bacteriological studies. One can make quite an accurate and dependable diagnosis by such methods even when the positive evidence of actually finding the tubercle bacilli is absent.

SUBMUCOUS ULCER

Let me invite your attention to one other urological problem which is quite common and very frequently unrecognized. **Submucous ulcers of the bladder** sometimes known as "elusive ulcer of Hunner" is not an uncommon condition. It is quite largely confined to women and occurs in mid life. The outstanding symptom is frequency, varying from a few minutes to an hour or so. It is practically the same day and night. There is marked urgency with a contracted bladder having a capacity of only one or two ounces. The bladder is very painful when full, in fact sufficiently so to demand immediate emptying and this oftentimes leads to incontinence. There is little variation of symptoms from day to day.

Unfortunately for the victim of this affliction, there are few coexisting signs or findings and the symptoms are all out of proportion to any demonstrable pathological lesions. There may be no abnormal constituents in the urine, such as pus, blood, albumin, etc., and it may even be negative to bacteriological culture. The x-ray findings and kidney function tests may also be normal and yet the symptoms are outstanding and always present. The woman is usually nervous and because of this and the negative findings she receives a diagnosis of a "nervous bladder." In these cases it is usually the bladder disability that makes the woman nervous and not the nervous woman that makes the irritable bladder. It is even sometimes difficult to make a diagnosis in such cases by a careful cystoscopic examination. The ulcers are not always visible. For this reason they have been called "elusive ulcers." Repeated efforts, however, are rewarded by findings of a localized reddened area with an irregular margin from which small distended vessels may be seen radiating out for a few millimeters. These cases can be relieved by the elimination of all focal points of infection, proper hygiene, particularly colon hygiene, along with over distension of the bladder under gas anaesthesia or fulguration of the ulcer. I find it profitable to keep these patients in bed for a week after the fulguration, with an indwelling catheter for drainage and the injection daily of one ounce of one per cent mercurochrome.

RESIDUAL URINE

The last urological problem I wish to discuss is that of residual urine,—its causes and effects. Residual urine, meaning the amount left in the bladder following micturation—when an effort was made to completely empty—may vary from one dram to sixty or more ounces and exist without attracting

serious attention to the bladder. Symptoms when present are not always in keeping with the amount of residual. A small quantity may be the cause of active symptoms; a large amount may be present without direct symptoms. Numbering among the conditions interfering with complete emptying of the bladder may be, obstructions at the bladder neck, such as enlargements of the prostate due to infections, hypertrophy and tumors, seminal vesiculitis, obstructions in the urethra, as calculi, strictures, polypi, papillomas, and atonic bladders secondary to a disturbance of the central circulatory nervous system such as tabes, syphilis, malformations, spina bifida, etc. Again this is not a complete list but it is given merely to show that when residual urine is demonstrated one should not attempt to explain its cause without making a thorough investigation. I wish to emphasize the importance and value of making routine efforts to determine the degree with which the bladder empties itself, as a part of all urological examinations. The technique is so simple and devoid of danger when the proper method is followed. Catheterization should be carefully and aseptically done immediately after voiding. The patient must be instructed to completely empty the bladder. The amount obtained should be collected in a sterile container in order to provide material for bacteriological study. After draining the residual the injection of one ounce of a five per cent solution of argyrol into the bladder serves two very useful purposes, prophylaxis against infection, and to determine the ability of the bladder to empty itself. The argyrol should be totally eliminated at the first micturation. The number of times it is manifest in after voidings presents positive evidence not only of residual but is an index to the degree of emptying. It further serves as a means of impressing the patient with the facts involved. There are two very important precautions which must be emphasized, a rigid aceptic technique, and never to drain off over eight ounces from a bladder retaining more than that amount. The cause of a given case of residual urine can usually be determined by simple bladder cystoscopy. Evidence of an obstructing prostate, diverticuli, incompetent orifices, etc., can as a rule be easily detected. There are cases though when the causes cannot be seen through the cystoscope. In such cases a cystogram offers great aid not only in presenting the evidence, or demonstrating the cause, but definitely determining the degree or extent of the lesion. Particularly is this true in incompetent ureteral orifices, small diverticula, etc. A plain plate should always be made before the injection of the opaque media in order to determine any orthopedic lesion of the lower spine, or any calculi in the urinary tract, should they exist. Thorough physical and neurological examinations are just as necessary in all of these cases as in any other obscure condition and by it the underlying cause of the patient's urinary symptoms can frequently be determined. The systemic effect produced by residual urine varies with the cause producing it. There seems to be more reflex effect upon the kidney function when an obstructing prostate is the cause. A much larger amount of residual urine caused by a diverticulum of the bladder may give rise to much less disturbance than an equal or less amount produced by an obstructing prostate. The most serious effect of residual urine is its predisposing factor to infection and to the formation of calculi. Any bladder which cannot empty itself at each micturation is quite prone to infection and stone. We serve the best interest of our patients by avoiding these complications which can easily be accomplished by discovering and eliminating the cause of the residual urine before any of these complications develop. It is to be remembered that the title of this paper is "Some urological problems" and this is not to be considered a discussion of all the urological problems of interest to general practitioners.

CONCLUSIONS

1. In conclusion, let me emphasize the importance of giving the urinary tract a thorough study when making the annual physical examinations of our patients.

2. To make such a study includes more than the simple examination of the urine, and palpation of the organs.

3. Symptoms are very unreliable and may be misleading and are not to be depended upon for a diagnosis.

4. While certain symptoms may be difficult to explain, the answer may be had by persistent and painstaking efforts.

5. Impending trouble discovered before it becomes imperative is more conservatively and successfully dealt with, than to wait until the condition becomes demanding.

6. One should always think of the urinary tract as a possible source of any pain which cannot be otherwise positively explained.

FACTORS OF SAFETY IN THYROID SURGERY

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(Read before the Southern California Medical Association, Santa Ana, April 4th, and 5th, 1930.)

It is not probable that the very large increase in surgical operations on the thyroid gland during the recent past represents an actual increase in the incidence of giotrous disease. Rather, it is to be interpreted as indicative of the rapid increment which has taken place with reference to surgery in general. True, surgical attack upon the thyroid is far more frequent now than formerly, but so is surgical attack upon every other organ and portion of the body. And this surgical-mindedness has been and is accompanied by conspicuous hazards and penalties. Instead of the decrease in mortality which would naturally be expected to attend larger experience, quite the reverse has occurred, because, undoubtedly, the experience has been distributed among such an increased number of different operators.

The mortality rate of all surgery is higher today than at any time within the past quarter of a century. This is notably true of thyroid surgery. Two decades ago it was generally accepted that this constituted a highly specialized field which only a relatively few cared to invade. As a result the mortality rate in the hands of those few steadily dropped from ten per cent to one per cent or less. Today, taken in the aggregate, it is again at or above ten per cent.

These are not pleasant thoughts. If true, they should give us serious pause. Surgery and the surgeon exist for the patient, not the patient for surgery and the surgeon. When this idea is fully assimilated one of the chief grounds of the criticism and censure from which our profession is suffering at the present time will be effectually removed.

One other preliminary observation. Anatomy is and must continue to be the essential basis of successful surgery. Intimate, exact, comprehensive knowledge of the regional anatomy of the neck is a fundamental requisite if these patients are to have a square deal at our hands. To remove a goiter is one thing. To remove it—just the amount it should be removed—without danger or damage to important contiguous structures is quite another story.

The factors which make for safety in thyroid surgery constitute a very large subject. Mention will be made here of only a few of them which seem of outstanding importance.

STUDY OF THE CASE

Undoubtedly this is the chief desideratum in these cases. The vital question is not to determine the presence of a goiter. Usually this is sufficiently obvious at first glance. What we seek first to know,—what the safety, always, and often the life of the patient depend upon,—are the history of the case, the care and completeness with which it is elicited and digested, and the thoroughness with which the physical examination is made. To be of real value all of these must include the patient as well as the goiter. What is the condition of the patient's general health? To what extent have the heart and other vital organs been impaired? What is the present degree of toxicity as indicated by a reliable basal metabolism test? Has the patient been taking iodine, and, if so, in what form and dosage, when and how long? Does the goiter cause pressure symptoms? Is it embarrassing by reason of its size? Is it definitely or probably substernal? Will the case be best handled by surgical intervention? If so, what length and what kind of preparatory treatment will be necessary? And so forth. These and many other questions of the utmost importance arise in every case. Until we have placed ourselves in position to answer them, all of them, in each instance we are not in position to justify the confidence patients repose in us when they seek our aid.

PREPARATION OF THE PATIENT

I do not hesitate to say that in the majority of cases the preparation of the patient is fully as important as the operation itself. Possibly more so, because the mechanical steps of a given surgical procedure may, by repetition, become largely matters of simple routine, while the subtle considerations involved in selecting the most favorable time for operation must be determined separately in every individual patient. Surgical judgment, that intangible factor which is so essential to successful surgery, plays a more vital part in preparing the patient and choosing the time to operate than does dextrous handling of scalpel and sutures.

One point in this connection should be especially emphasized, namely, that the time spent in preparatory treatment is not often wasted. Generally speaking, there is nothing to be gained by haste. Far better delay a few days longer than absolutely necessary than to hurry into operation even a single day before the most propitious time has arrived.

Every surgeon who handles these cases at all frequently is likely, rather early in his experience, to adopt certain rules to guide him in deciding when it is safe to op-

erate. Of course I mean principles rather than inflexible rules. Personally, I confess to a decided reluctance to proceed with the operation until the basal metabolism reading has been reduced below plus fifty and the pulse rate below one hundred. To bring about these favorable conditions sometimes requires much patience on the part of all concerned; but the time so spent is seldom a real loss and the added sense of security more than compensates for the delay.

CHOICE OF ANESTHETIC

Much of the foregoing consists of generalities. Such propositions as "study of the patient" can not be reduced to definite formula. Individualization in each and every case is the only means by which the patient's welfare may be effectually safeguarded.

Choice of anesthetic is a question that permits and invites a more concrete discussion. And there is much that should be said upon the subject, for the outcome of the operation is always at stake, and especially so in hyperthyroid cases.

In general terms we may say that two very different types of cases are encountered, the simple or non-toxic, and the hyperthyroid or toxic cases. In the former, my personal preference is local anesthesia alone, and I find myself employing it to a constantly increasing extent. It is true that the method makes many exacting demands in technic and finesse; but there is a degree of satisfaction attending its use which far overbalances any incidental difficulty or disadvantage.

The conditions are radically different in the toxic cases. Here the extreme mental alertness and the keyed-up emotional state render the patient peculiarly susceptible to the evil effects of external stimuli of every kind. Mere contemplation of the approaching ordeal is sufficient to cause grave disturbance, which the sights and sounds of the operating room serve to exaggerate tremendously. There can be no doubt that the danger and the mortality rate are definitely increased by these factors.

It admits of no question that the ideal way to manage the hyperthyroid case is to protect the patient from these untoward influences by every means in our power. Crile recognized this truth years ago and his so-called anoci-association method was the outcome. Careful observation of the details of this method renders it possible literally to "steal the goiter." Unfortunately, few hospitals afford the necessary facilities for carrying out this ingenious plan and most of us can take advantage of it only by resorting to expedients which in reality are merely makeshifts.

The introduction of the new drug, sodium amytal, has done much to improve the situation and make it possible for all of us to utilize the principles involved in the Crile method. By the proper use of this agent patients may be tided over the entire operative procedure with a minimum of disturbance and without inducing harmful reactions of any kind.

Sodium amytal is a hypnotic. Within the limits of safe dosage it is not an anesthetic nor an analgesic. This fact should be kept constantly in mind. The only evil effects reported which could fairly be attributed to the remedy—and the cases are surprisingly few in number—have occurred in instances where injudicious doses were employed.

The drug is given intravenously only. The amount required varies from seven to fifteen grains. It acts with remarkable promptness, the patient falling into a profound and apparently natural sleep while it is being administered. This is the indication that enough has been given. Nothing more than a state of unconsciousness should be expected or sought. The sleeping patient may be transported to the operating room, the supplementary anesthetic, local or general, started and the operation completed without the patient knowing that anything has occurred until she awakens in her accustomed surroundings some hours later and inquires what it is all about.

My experience with the method described embraces some twelve or fifteen cases, all within the past nine months, in none of which were observed any alarming or even disquieting effects properly chargeable to the sodium amytal. After a moderate temporary fall the blood pressure remains unchanged and the pulse rarely rises more than ten beats throughout the entire operation.

I am impressed that as a rule it is better to supplement the sodium amytal with general than with local anesthesia. Much less of the nitrous oxide or ethylene will be found necessary. Ether I do not advocate for this or any other purpose when a competent anesthetist is available. The objection to local anesthesia as an auxiliary is based on the fact that the cooperation of the sleeping patient can not be enlisted and, unless the surgeon is especially skilled in the technic of the local method, disconcerting reflex movements and restlessness on the part of the patient are apt to occasion both delay and embarrassment.

Finally, in this connection let me strongly recommend that the administration of

this new agent be invariably in the hands of a trained anesthetist. Properly considered it is not a part of the surgeon's job. Personally, I should no more think of resorting to it without the services of the anesthetist than of undertaking a thyroidectomy without an adequate supply of artery forceps.

THE OPERATION ITSELF

As suggested in my introductory remarks, thyroidectomy is not a simple operation. A surgeon friend said to me only a few days ago that he could not understand why any sane man would choose to specialize in this field; that the numerous problems involved were too serious and intricate for comfort; and that he much preferred, both for his own sake and for the patient's to remove a gallbladder, or an appendix, or a uterus, because when he was through with one of these procedures he could be sure that it was finished and that he had done only the correct things. A vast amount of important truth underlies those frank observations. To know what and how much to remove in a thyroidectomy are questions no more weighty in their import than to know what and how much not to remove.

Lest I be charged with presumption, I shall not enter into discussion of the details of surgical technic. Instead, let me conclude by simply mentioning a few salient points:

1. Operation on the thyroid gland should always be approached with a keen sense of responsibility and a full appreciation of every tissue and structure in the neck, realizing that it is as important to conserve certain of them as it is to attack certain others.

2. Gentleness of manipulation is peculiarly essential in this work. The amount of traumatism inflicted is always reflected in the patient's condition both during and at the termination of the operation.

3. Speed but not haste is highly desirable. Excessive time consumed in an operation is also apparent in the patient's condition when the ordeal is over.

4. Hemostasis should be complete at all stages of the operation. Undue loss of blood adds danger and days to the convalescence.

5. I am firmly convinced that, with rare exceptions, provision for drainage should be made in these cases. When it is omitted there is apt to be considerable rise of temperature during the first week and the wound will often have to be opened for this purpose.

6. There is no operation in the entire range of surgery which is attended by such speedy and spectacular benefit to the patient. This is often noticeable from the first day in the tempered heart action and more tranquil facial expression. More than this, it is not infrequently as truly a life saving procedure as the removal of a gangrenous appendix.

AN ATYPICAL CASE OF NEUROSYPHILIS WITH NEGATIVE SEROLOGY.

R. C. KIRKWOOD, M. D.
Medical Director, Apache Powder Company, Benson, Arizona

F. C., male. Mexican, age 23, married, employed by Apache Powder Co. during the past 11 years, was referred to me by the company, June 24, 1930, because of difficulty in walking.

Family history. Father died at 47 years of unknown cause, after a febrile illness of several weeks duration; had always been healthy, was not given to the excessive use of alcohol. Mother living and well, aged 45, resides in Sonira, Mexico. No history of alcoholism, epilepsy or other nervous or any mental disease in family. Wife and one child, aged two years, living and well. One child died of unknown cause when 25 hours old. Wife has not had any miscarriages.

Personal history. Born in Old Mexico. Patient insists he has never had any of the acute infections of childhood, and that he has never had any serious illness, operation or injury. Except for a rare headache or a "cold" of a day or two s duration has never been ill and, in the more than 11 years employed by the company, he never consulted the company physician until the onset of present illness, June 24, 1930. Patient denies veneral disease; states he has never used alcohol to the point of intoxication and never regularly. Denies the use of narcotics or hypnotics or any drug at any time.

Present illness. Patient insists the onset was sudden, that he never noted any difficulty in walking until June 23, 1930, the day before he reported to the company physician, but reliable witnesses who have known patient for years, state they have noted a peculiarity in his gait becoming gradually more marked over a period of one and a half to two years. Patient's chief complaint was difficulty in walking interfering with the performance of his usual work, a difficulty which he insisted was due not to weakness but to lack of co-ordination of his limbs. Patient says, he has, and has had, no headaches, diplopia, dizziness, palpitation, dyspnea, dysphagia, jaundice, tinnitus, otitis, tonsillitis or sinus disease, no cough, expectoration, hemoptysis or pluerisy, no abdominal pain or tenderness, no diarrheal diseases. Appetite and digestion good. Bowels regular; vision normal; hearing unimpaired; no urinary disturbance; no edema.

There is a negative history suggesting syphilis, possibly hereditary.

Physical examination. Patient is a fairly well developed, moderately well nourished, adult, male Mexican. Mucosa appears somewhat pale. Skin shows scattered areas of vitiligo, varying in size, over entire body, which he says he has had as long as he can remember. There are no trophic changes in the skin and no scars suggestive of a healed primary lesion, no pigmentation suggesting healed secondary eruption, no mucous patches.

There is no general adenopathy; thyroid is not enlarged; there is no abnormal sweating, no tache or other evidence of pathology of the ductless glands or of the vegetative nervous system. Heart and lungs appear normal. There is no arteriosclerosis. Blood pressure is systolic, 94-100; diastolic, 60-64. Genito-urinary system, negative. Repeated urinaly-

sis shows amount normal; urine amber, clear; acidity normal; sp. gr., 1014 to 1022; no albumin; no sugar; no acetone; no casts or cylindroids. Gastro-intestinal tract negative; no masses; no scars, liver and spleen not palatable. Extremities normal; no flat-foot, no deformities, no muscular atrophy or hypertrophy; mobility of all joints unimpaired.

Neurologic examination. Cranial Nerves No. 1: No evidence of disturbance of sense of smell. No. 2: Vision unimpaired, fields normal, no hemianopsia; Fundi and retinae normal; no lesions of blood vessels; nerve head distinct in outline, appears normal. Nos. 3. 4 and 6: No. ptosis; ocular movements good in all directions; no squint; no nystagmus; pupils equal, round, contracted, react perfectly to accomodation but very sluggishly to light, and only to the very brightest, most concentrated light at all; to the light of a flashlight or opthalmoscope no reaction occurs. No. 5:No disturbance of sensation of face; masseters and temporals contract normally and equally. No. 7: Face symmetrical; no impairment of motility except that the left side of mouth is drawn a litle farther outward when showing teeth with jaws together. No. 8:Hearing unimpaired; no tinnitus or vertigo; fork tests normal. Nos. 9 and 10: Swallowing not impaired; palate moves evenly; sense of taste unimpaired; vocal cords show no pathology, their function unimpaired. No. 11: Sterno-mastoids and trapezii contract strongly and equally. No. 12: No atrophy of tongue, no tremor: tongue protrudes centrally and evenly.

Motor System. Station decidedly impaired, tends to stand upon a wide base with feet apart. Romberg markedly positive. Standing on one foot with eyes open, shows marked impairment of equilibrium. Patient walks with feet wide apart, with very definite uncertainty, constantly looking down at his feet; turns quickly and with difficulty—a very typical ataxia. There is no endency to fall in any particular direction, no propulsion or retropulsion. No tremor, no athetosis, no twitching or rythmic movements. No loss of power in any group of muscles. Coordination markedly impaired in arms and hands, as well as feet and legs, by all usual tests. Incoordination in heel-to-knee tests, more marked on right side. No adiadokokinesis.

Knee jerks diminished when first seen, later apparently equal and adequate. Jaw, palatal, abdominal, cremasteric and plantr reflexes present. Biceps, rticeps and radius reflexes absent. Achilles reflexes present and equal. There is no clonus, Babinski, Gordon or Oppenheim. Micturition and defecation unimpaired.

Sensory System. No subjective disturbances. No impairment of pain or temperature sense. Vibration sense appears to be less acute in left leg than in right. No tenderness of peripheral nerves. Position sense poor in both arms and legs. Pinching Achilles tendon produces normal pain. Patient differentiates between knife and pencil placed in his hand, with eyes closed, but is slightly astereognostic for coins. mistaking a silver dollar for a quarter in left hand. and a dollar for a half dollar in right hand.

Mental Examination. Essentially negative. Well oriented for time, place and people; insight good: general and special memory good for one who had only a few years in school; no illusions or delusion no hallucinations (visual or auditory); response to ethical questions perfect. Patient rather introspective and appears to enjoy being examined by so many physicians and by so many methods.

Laboratory Findings. Blood and spinal fluid Wassermann and Kahn tests repeatedly negative at three laboratories. Colloidal gold curve normal Spinal fluid not under pressure, cells 3 to 7; Serum globulin, none at any test in any laboratory.

Blood shows 4,500,000 to 5.200,000 R. B. C.,; hem. 55 per cent to 75 per cent; 6,500 to 7,800 W. B. C.; differential count normal.

Urine: lemon to amber, clear, acidity normal, no sugar, no acetone, no cases at many examinations.

X-ray shows no pathology of hip or pelvic joints, nor of the bones entering into these joins or of the long bones of the lower limbs.

Diagnosis: Neuro-syphilis, tabes dorsalis.

DISCUSSION

The case presents the following points:

1. Ataxia of sensory type;
2. Marked Romberg;
3. Argyll Robertson pupil-reaction only to very brilliant light and that very sluggish;
4. Incoordination of arms and legs;
5. Absent deep reflexes in arms, diminished in legs (knee jerks);
6. Impaired position sense in arms and legs;
7. Slight astereognosis;
8. Spinal fluid and blood, negative.

The symptoms and neurological findings all point conclusively to a degenerative process in the posterior columns of the spinal cord (columns of Goll and Burdock) in a relatively early stage, with the finding of an early Argyll-Robertson pupil. These findings are those common to neurosyphilis of the tabetic type. The sudden onset claimed by the patient, but disproved by other observers, suggests the possibility of multiple sclerosis; but the absence of confirmatory evidence—especially the presence of abdominal reflexes, the absence of intention tremor, of speech disturbance and of nystagmus—makes this diagnosis untenable. The negative blood and spinal fluid appears to militate against neurosyphilis but it must be remembered that seven per cent of very definite tabetics studied by Kaplan were found to be serologically negative, and this case is apparently one of that seven per cent.

The question of etiology has been of especial interest in this case, in that the suggestion was made that, the man having been employed in the manufacture of explosives for more than eleven years, his symptoms might be the result of his constant exposure to sodium nitrate or the dust or fumes thereof. However, study of the effects of exposure to this chemical prove that, when it causes symptoms at all (which is extremely rare) it is a respiratory irritant, the symptoms being cough, expectoration and even hemoptysis, but in no case has it caused any damage to the central nervous system. Other employees of the Apache Powder Company exposed to sodium nitrate as long or longer than the patient, have never developed similar or any symptoms. Replies from the medical directors of E. I. duPont, deNe-

mours & Co., of Hercules Powder Co., are to the effect that in a very large experience covering many years in the manufacture of explosives, employing many men, no symptoms have been encountered which could be ascribed to exposure to sodium nitrate. The office of the Chief of Chemical Warfare Service, U. S. Army, reports no such symptoms have ever been reported from exposure to this substance (sodium nitrate) and the Library of the Surgeon General, U. S. Army, reports no cases of record in the literature of any symptoms referable to the central nervous system due to sodium nitrate. Therefore, the question of exposure to sodium nitrate as the cause of the symptoms which this case presents, can be dismissed as impossible.

The diagnosis stated above—neurosyphilis, tabes dorsalis—has been concurred in by Dr. E. A. Duncan, El Paso, Texas, and Dr. N. C. Bledsoe, Bisbee, Ariz., after careful study of the case and observation in hospital.

The case is of interest because of the definite clinical picture it presents in spite of the entire lack of serological confirmation.

TREATMENT

Patient has been on what in the past has been called "mixed treatment", mercury and iodides orally without notable effect as yet, except possibly some slight improvement in general condition, notably increase in percentage of hemoglobin. At present he is being intensively treated with intravenous neo-salvarsan and intramuscular injections of bismuth salicylate, gr. 2, per ampule.

PROGNOSIS

Guarded; the symptoms presented may become progressively worse in spite of the active treatment he is now receiving.

DIAGNOSTIC OPEN FORUM

I. CASE OF ORTHOPNEA

(No. 15351, Case Records of the Mass. Genl. Hosp., from N. E. J. of Med., Aug. 29, 1929, p. 424.)

Case Record

A Portuguese Negro from the Cape Verde Islands fifty-six years old entered January 30 complaining of orthopnea of ten days' duration. The history, brief as it is, had to be obtained by leading questions because of his orthopnea, discomfort, language difficulty and lack of intelligence.

For a month before admission he had had increasing dyspnea. Medicines given by his physician gave no relief. Ten days before admission he began to have cough with half a cupful of white frothy sputum a day. For a week he had had palpitation, a feeling of drowsiness, and anorexia due to epigastric pain and fullness whenever he ate or drank, and had not been able to lie down, but had slept in a chair. Once he vomited. For a week his urinary output had been small. Recently he had noticed a tightness in his chest. The day of admission he noticed flecks of blood in the sputum.

He had always been strong. Like everyone else in the Cape Verde Islands, he had had many fevers lasting two or three days. He had been in Massachusetts for eighteen years. Thirteen years before admission he had a penile lesion that cleared up in a week. He denied all other symptoms of venereal disease. For seven months he had urinated once at night. His bowels were constipated.

Clinical examination showed a well nourished man, very dyspneic and orthopneic. Teeth bad. Marked pyorrhea. Apex impulse of the heart not seen or felt. Left border of dullness 10 centimeters from midsternum, 2 centimeters outside the midclavicular line, right border 3 centimeters, supracardiac dullness 5.5 centimeters. At the apex was a soft systolic murmur and a loud early diastolic; at the base a louder systolic and the same early diastolic heard also along the left border of the sternum. Rhythm regular. Sounds of good quality. Pulses normal. Artery walls palpable and tortuous. Blood pressure 140/50. An electrocardiogram showed normal rhythm, rate 100, rather low T waves. Expansion of the right lung greater than the left. Crepitant rales throughout the left lower lobe posteriorly. Liver dullness increased downward below the costal margin. Tender right upper quadrant. The patient resisted palpation here. There was a penile scar. Prostate large, tender, symmetrical. Edema of the legs to midleg.

Urine: 32 to 46 ounces, specific gravity 1.029 to 1.030, urine cloudy at all of three examinations, the slightest possible trace of albumin once, 2 to 3 leucocytes in one of three sediment examinations. Renal function 50 per cent. Blood: 17,900 to 11,700 leukocytes, 85 per cent polymorphonuclears, hemoglobin 80 per cent, reds 4,620,000, smear normal. Two Hinton and two Wassermann tests strongly positive.

Tempertaure 96.6° to 99.7°. Pulse 80 to 100, with a terminal increase to 128. Respirations 20 to 32, with a terminal increase to 43.

X-ray examination showed the size and shape of the heart shadow to be within normal limits. The aorta was not increased in its transverse diameter. It was a little wide across the auricle. There was rather coarse mottled dullness occupying the greater portion of both lung fields. The changes were more marked around the root and gradually faded out toward the periphery. They obscured the outline of the heart and the aorta. The diaphragm was visible on both sides. Its respiratory movements were limited. On the left the costophrenic sinus was distinct. The examination did not show any positive evidence of luetic aortitis or enlargement of the heart. There was probably some dilatation of the auricles.

Under digitalis the edema disappeared and for a few days the patient improved. February 5 and 6 he was very delirious and thought he was being killed. A psychiatrist thought the delirium was associated with the physical disease and might be transitory. February 6 the patient was very dyspneic all day. That night he suddenly died in a fit of anger.

DISCUSSION

DR. W. WARNER WATKINS

(Before Phoenix Clinical Club.)

I like the plan of a four step approach to a diagnosis. First on the basis of history of complaint and past history, to list the diagnostic possibilities; second, on the basis of physical and clinical findings, to eliminate some or add other possibilities; third, on the basis of the various laboratory findings,—clinical and x-ray,—to correct still fur-

ther the diagnostic suggestions; and fourth, to select the preferable diagnosis by deductive differentiation.

(1) This man's personal history suggests tropical disease or syphilis. His residence of 18 years in Massachusetts dims the possibility of tropical disease, but the penile lesion 13 years ago keeps syhpilis in the forefront. The chief complaint was dyspnea, which can be caused by any condition which interferes with circulation and respiration. The dyspnea began at least a month before admission and for the past ten days has been severe (orthopnea). The possibilities in order of preference at this point ,would be:—

Heart disease with decompensation, including aortitis and aneurism.

Lung disease, with or without emphysema or pneumothorax.

Pleural or pericardial effusion.

Newgrowth in mediastinum or lung.

Abdominal disease crowding on the chest.

(2) We make the physical examination and, to our surprise we do not find the heart much enlarged; we find pulses normal and regular, rate 80 to 100, pulse pressure slightly increased, a very suggestive heart murmur, diminished expansion of left lung and rales in lower left chest, edema of the legs, respirations 20 to 32, slight fever. The murmur is a soft systolic and loud diastolic, heard at apex and base and transmitted along sternum; liver dullness increased, tenderness in upper right quadrant, prostate enlarged and tender. These are paradoxical findings, and about all they do with positiveness is to rule out abdominal disease crowding on the chest sufficiently to produce the orthopnea. They still leave us with possible diagnoses.

Heart disease with decompensation, although with paradoxical findings for this.

Lung disease, including mediastinal or pulmonary newgrowths.

Pleural or pericardial effusions almost though not absolutely ruled out. Failure to feel the apex beat and limitation of expansion of left lung keep these within the field of possibilities.

We incline, very strongly, still, toward heart disease, and would require x-ray findings with regard to heart size, shape and muscular action, and the shadows in the lungs, with other laboratory data. In fact, we feel, at this point, that all the help we can get will be welcome.

(3) The laboratory gives us normal urine and normal blood counts except for leucocytosis. Two positve Wassermanns, on top of our definite history of penile lesion 13 years ago keep syphilis in the forefront. The x-ray of heart shows normal size and shape, without increase in aortic shadow but with some evidence of dilated auricles. There is a coarse mottling in the lung fields, occupying the greater portions of both sides, gradually shading off toward the periphery, but obscuring the heart and aortic outlines in the perihilar areas. This gives us something to think about. What do the laboratory data do to our diagnostic possibilities? They firmly fix syphilis into the diagnostic pattern. They rule out pleural and pericardial effusion. They throw grave doubt on the presence of cardiac decompensation as a cause of the dyspnea. They give us some lung lesions to be explained. In other words, they leave us two groups of lesions,—heart and lung,—to be considered, but with some new and troublesome diagnostic data to be woven into the differential pattern. We have now to answer the question, What can cause a marked dyspnea, with the diastolic murmur but without increase in size of hert, yet with circulatory obstruction and passive congestion in abdomen, and edema of legs, leucocytosis, and sudden death?

(4) We enter the fourth step of differentiation, stubbornly adhering to our predilection for heart disease, but feeling very shaky in the position.

A diastolic murmur ought to mean valve disease, and when heard at base, apex and along sternum, should mean aortic insufficiency, but we have nothing but the murmur, the dyspnea and passive congestion to support this. Without enlargement of the heart, without a Corrigan pulse, with sounds of good quality, rythm regular pulses normal to feel, pulse pressure only slightly increased, with flat T waves in electrocardiogram, we cannot say decompensation, and if we do not have decompensation, we cannot explain the dyspnea on the basis of aortic incompetence. In the face of paradoxical findings like these, we would reject the diastolic murmur before we would the other findings.

If not aortic valve disease, what else can cause a diastolic murmur? Chronic mitral disease may give the Graham-Steele murmur at times. Could we have mitral stenosis which would keep the heart small, and an aortic disease which would attempt to make it large, and have them offset each other, so that we have a normal size heart? The auricular enlargement would be compatible with mitral disease. This is a fanciful diagnosis, and if we can find other explanation for the symptoms, we should not make it.

Yet, if we are going to diagnose valvular heart disease, we must do it in the face of physical findings not in accord with the pathology in question. We must either diagnose syphilitic disease of the aortic valve with hypertrophy and decompensation, in spite of physical and x-ray findings. Or we must diagnose some rarer heart lesion or combination of lesions, producing circulatory failure without ventricular enlargement,—at least of the left ventricle. Before deciding the question finally, let us consider the pulmonary causes of dyspnea.

If we can get over, under or around this diastolic murmur, we can explain the other symptoms by lung disease.

Miliary tuberculosis or other infectious process in the lungs, with or without emphysema would explain the dyspnea, the circulatory embarassment. We have little support for this group, outside of the x-ray findings which would not be in accord with miliary tuberculosis.

I thoroughly agree that the x-ray findings must be subordinated to the clinical picture and not dominate it. Therefore, in the absence of fever, emaciation or other symptoms of miliary tuberculosis, I pass that by.

For pulmonary newgrowth, we have more basis. We have a coarse mottled dullness through both lung fields, more marked in the perihilar areas, where they obscure the outline of heart and aorta, and diaphragm is limited in mobility. We could not attach great importance to such lung shadows if we had a decompensating heart or circulatory stasis from heart disease, but if we do not have these, the lung shadows are important. Are they the shadows of newgrowth, and if so, are they primary or metastatic? They can be either, and by pressure could obstruct circulation through lungs or return flow from lower half of body.

One other possibility which would be in the same category as newgrowth suggests itself to me, and that is a syphilitic involvement of lung hilus, extending into lung tissues, and causing mechanical pressure on large vessels about the base of heart. Along with this we could have a slight degree of syphilitic involvement of aortic valve enough to cause the murmur. Or, conceivably, we might have

fibrotic changes which would so deform the structures as to give us a relative insufficiency of one or more valves.

I am going to pin my faith on my x-ray confrere, whoever he may be, and say, no cardiac enlargement, therefore no hypertrophy or decompensation; therefore the dyspnea was not due to valvular heart disease. The dyspnea and circulatory disturbances were due to lung disease, preferably carcinoma of the lungs,—whether metastatic or primary, we will not attempt to say. We believe there will be syphilitic arotitis, causing enough relative aortic incompetence to give us a murmur, with cardiac infarct as the immediate cause of death.

Furthermore, we are willing to bet four bits that this diagnosis is wrong.

DR. RICHARD C. CABOT

(In part, from journal mentioned.)

The history of the present illness all tends to make us think of a heart lesion back of it all, with passive congestion of the different parts of his body.

I suppose the fevers were malaria or unidentified types of infection.

Those percussion measurements are all within normal limits for an adult man. We have no proof so far of cardiac enlargement.

The essential thing in the blood pressure is the pulse pressure of 90, which of course is increased.

From the electrocardiogram, on the basis of a low T wave with nothing else, I do not think anyone would venture to say anything in particular.

The penile scar does not in the least prove that he had had syphilis. Ducrey's infection, chancroid, leaves scars more often than syphilis.

The urine is normal. The blood is essentially normal until we come to the Hinton and Wassermann tests.

It is rather surprising that x-ray showed the size and shape of the heart shadow within normal limits, because we have a diastolic murmur and presumably aortic regurgitaiton, and in most cases the ventricle is hypertrophied. I should not be surprised if it was hypertrophied here, and the heart does weigh more even if it has not stretched,—the concentric type of hypertensive heart with very little dilatation. Hypertrophy-and-dilatation usually is thought of as all one word. Now and then we find hypertrophy withtout dilatation.

We have no evidence of aneurysm from the x-ray.

The signs in the lung fields are presumably due to passive congestion, perhaps to bronchouneumonia. We cannot tell from the x-ray. Such changes around the root make you think of passive congestion.

I suppose the costophrenic sinus was indistinct on the other side, presumably from fluid.

I do not believe x-ray can make the diagnosis of luetic aortitis unless there is aneurysm.

The psychiatrist did not think the patient had any of the regular types of insanity.

DIFFERENTIAL DIAGNOSIS

What are the essential facts in this case? A man of fifty-six, a Negro, always well except for possible syphilitic infection,—we do not know about that—and for fevers which we cannot believe had any permanent effect upon him. He seems to have dyspnea, orthopnea, passive congestion; probably has heart disease. We listen to the heart and find a diastolic murmur and nothing else. That is enough to make a diagnosis of aortic regurgitation, especially when backed up by a large pulse pressure such as there is in this case. The rest of the physical examination shows positive Wassermann and Hinton tests and passive congestion, probably shown in the liver, the lungs and the legs. The diagnosis is syphilitic heart disease and aortic insufficiency.

What symptom that he did not have here would you expect if it is infarct of the heart? He ought to have had pain. Dr. Mallory has shown us cases of coronary occlusion without pain, so that does not exclude occlusion of the mouth of the coronary arteries where the syphilitic process is. It may have been the cause of death. It does not seem to me that we have enough passive congestion to account for death. His legs were not much swollen and his lungs were only moderately congested.

Coronary occlusion is as good a suggestion as I can make as to the cause of death. I do not believe there was a brain lesion. There may be a terminal infection, bronchopneumonia most probably, which would account for the delirium and perhaps account for death. My diagnosis is syphilitic aortitis with aortic regurgitation; then possibly occlusion of the coronary arteries and sudden death from that cause, or very possibly bronchopneumonia and death from that cause.

DR. TRACY B. MALLORY

Anatomic Diagnosis: Syphilitic heart disease, syphilitic oartitis, bacterial endocraditis.

This man had syphilitic heart disease. The heart was hypertrophied somewhat, weighing over 500 grams. There was definite insufficiency of the aortic valve. There was also a terminal infection, which however was a somewhat surprising one in that it was bacterial endocarditis, which in my experience is relatively uncommon as a sequel of syphilitic heart disease. The majority of cases of subacute bacterial endocarditis infection show an antecedent rheumatic infection, although congenital lesions not infrequently are predisposing factors.

All the other organs were essentially negative. The aorta showed some evidence of syphilis, but nowhere enough dilatation to cause aneurysm. There was an anomaly of the liver, probably congenital, of no significance.

Dr. Cabot: One point of interest is that you can have a heart as big as this, 500 grams, and x-ray does not show it.

A Physician:; Did you find anything to account for the sudden death?

Dr. Mallory: No, I did not, except that the acute bacterial endocarditis unquestionably played a considerable part in the picture, and the heart was probably in much worse shape than the mere symptoms of decompensation suggestetd.

Dr. Cabot I do not see that we can blame ourselves for not suspecting bacterial endocarditis. You can suspect it only when you have heart murmurs not otherwise accounted for, or embolism, or a positive blood culture. We had no evidence of embolism, no positive blood culture. We cannot say we were wrong because we forgot anything or reasoned wrong.

CASE OF DROWSINESS AND NOSE-BLEED

(No. 13581, Case Records of Mass. Genl. Hosp., from Boston M. & S. Jour., of Feb. 9, 1928, p. 1521.)

Case Record

First admission. A Canadian rubber worker twenty-eight years old was sent from the Out-Patient Genito-Urinary Department August 31 for swelling and tenderness of the left testicle of three months' duration. The swelling increased for two months after its appearance, then decreased. At admission he still had a hard tumor, and a discharging sinus on the right testicle.

His past history was entirely negative so far as he could remember except for a running sore on the left testicle four years before admission.

The family history is not important.

Clinical examination showed enlarged axillary and inguinal lymph nodes varying in size from being just palpable to the size of a grape. In the right groin the glands below Poupart's were enlarged and slightly tender. The epididymis was much thickened, indurated, adherent to the skin and showed a small crater-like sinus with rather abrupt edges. The upper pole of the left testicle was thickened.

At operation a much thickened and in places caseous epididymis was removed. A pathological report of tuberculosis was made. The patient made a good convalescence and was discharged relieved September 11.

History of interval. He was given tuberculin in the Out-Patient Department for two years and a half. At the end of that time he was feeling perfectly well, with no symptoms. The wound was clean and solid. In September, three years later, examination in the Accident Room showed no masses. The left epididymis contained a few small hard nodules which were not increasing in size. The general condition was excellent—appetite good, weight increasing, no cough. Five years after this note he had a right nephrectomy in another hospital following a kick from a horse. There was "blood clot" in the kidney(?). (Tuberculous kidney?) He was out of work six months. After this he was free from symptoms for five years. Two years before admission he had nycturia three or four times for four months. The same year he had a "pimple" in the penis which started to spread and located on the foreskin. He treated it with a salve which made it break and spread. It did not clear up for eight months. A year before admission he felt tired and run down. He seemed to improve after a short time, but in about ten months again began to tire easily, especially when doing heavy work, although he could run without any ill effects. About a month before his readmission he began to be dyspneic on climbing one flight of stairs, and felt drowsy. He grew gradually worse. A physician put him on a diet without relief. For four days before readmission he vomited about once a day. He thought his urinary stream was not so strong as it had been. There were no other urinary smptoms. For four days he had been in bed for the first time. For two days before readmission he had been unable to bear his weight on his legs. The day before readmission he could not move his hands at all. The day of admission he could move them a little. He had a tingling feeilng like pins and needles in his legs and feet. His feet were cold and numb. He felt drowsy. For seventy-two hours (?) he had not slept. The day before admission and the morning of admission he had nosebleed for a short time. His mouth was dry. He thought he had had no loss of weight during the present illness. His best weight was 160 pounds, when he was nineteen years old, his present weight 137.

No benzol was used in his department of the factory and he did not come into contact with it in other departments.

Second admission, August 27, fifteen years after his first admission.

Clinical examination showed a pale, poorly nourished man lying flat in bed in no apparent distress, with bleeding from both nostrils and dried blood upon his lips. The skin and mucous mmebranes were pale, with a questionable slightly icteric tint. The eyes showed subconjunctival hemorrhage about the outer circumference of the iris and across the sclera at two o'clock. On the under side of the tongue were two moist lesions, not indurated, one centimeter and half a centimeter in diameter. The few remaining teeth were decayed. There was bleeding about the roots of all the lower teeth. The apex impulse of the heart was not found. There was no enlargement to percussion. The sounds and action were normal. A blowing systolic murmur replaced the first sound at the apex and the base. The artery walls were normal. The blood pressure was 155/100. The abdomen showed voluntary spasm. In the left upper quadrant was a mass descending with respiration, feeling more like kidney or glands than spleen. There was resistance and dullness in the right upper quadrant, perhaps voluntary; no mass was felt. On the dorsum of the penis was a lesion 3 by 1.5 centimeters, indurated along the upper border, punched out, old in appearance, oozing serum from the center. The legs showed weakness but not paralysis. The arms showed slight weakness. The pupils and fundi was no remal, the knee-jerks diminished. There was no reaction on stroking the foot for Babinski. The touch sense was diminished. The other sensory reactions were normal.

The amount of urine is not recorded. A specimen in the Emergency Ward was loaded with pus. An overnight specimen showed 0 to 1 leucocyte per field, no red cells, a very slight trace of albumin, specific gravity 1:016. Blood: 5,800 leucocytes, 88 per cent. polynuclears, hemoglobin 25 per cent., reds 3,000,000, moderate achromia and variation in size and shape, rare polychromatophilia, reticulated cells 2 per cent., platelets increased. Wassermann negative.

Temperature 97° to 99°, rectal. Pulse and respirations normal.

The patient entered at four o'clock in the afternoon. He seemed semistuporous and was anxious to be put to sleep. Veronal was not effectual, codein was vomited and paraldehyde by rectum was expelled. At midnight 1/6 of a grain of morphia and 1/200 grain of scopolamin were given subcutaneously. At one o'clock in the morning there was complete paralysis of the legs, with absent reflexes and rapidly progressing paralysis of the arms. Respiratory paralysis soon set in. A lumbar puncture was negative. At three o'clock the patient died from steadily increasing respiratory paralysis.

DISCUSSION

(Before Clniical Club of Phoenix, Mch. 24, 1930.)

DR. JOSEPH M. GREER

The first thing that strikes our attention in this case is the swelling and tenderness of the testicle of long duration. The length of time almost rules out an epididymitis of gonorrheal origin. The next two things to consider are tuberculosis and malignancy. The tenderness points toward tuberculosis rather than malignancy. The observation that the swelling decreased after two months points against a malignancy and the discharging sinus is in favor of tuberculosis. The running sore on the testicle four years before is suspicious of tuberculosis.

The enlarged glands in both axillary and inguinal regions makes one think of some of the other conditions that produce adenopathy, such as Hodgkin's disease and the leukemias. In Hodgkin's disease, however, the enlargement is usually unilateral, at first at any rate, and is noted first in the submaxillary and cervical glnads. The leukemias may be ruled out by the blood picture. The general adenopathy is not the picture of glandular tuberculosis or the leukemias and it is probable that we shall have to look elsewhere for its cause. The patient's general condition would no doubt produce some modcrate adenopathy of these regions.

The thickened and indurated and adherent epi-

didymis, clinically is tuberculosis and we note at operation that a caseous epididymis was removed and the pathologist reported tuberculosis. Therefore so far may make a diagnosis of Tuberculous Epididymitis.

The interval history with improvement is rather characteristic of tuberculosis. As to the diagnosis and reason for nephrectomy five years later following injury we can only guess, as the history is silent upon the length of time after injury that the kidney was removed. The "blood clot" could be either injury or tuberculosis. It would be my guess that it was tuberculosis.

The next sign, namely, pimple on the penis, opens up the great question of syphilis and we will no doubt need some help from Dr. Colhessy on this subject. It is my opinion, however, that it was not syphilis for upon this alone in a patient with this history with no history of secondary manifestations and no history of antiluetic treatment and a negative Wassermann it would seem that we would not be justified in a diagnosis of chancre. It is possible that the condition was only a herpes and its chronicity due to over treatment.

The tire easily symptom suggests tuberculosis, although his anemia might do it. We would like to know something about the chest, but the history and physical findings are absent and the usually present radiologist on a vacation. We must think of his anemia which may account for his fatigue. This is no doubt a secondary anemia and accounts for his paleness icteric tint and heart murmur. It may also account for his present hemorrhages from the nose and gums. Hemophilia probably should be kept in mind, but we have nothing in either the history or the laboratory findings to suggest it. The mass in the abdomen was probably an enlarged kidney, maybe a pyonephrosis as one specimen was loaded with pus and one was not. We might think of a hypernephroma but there is no mention of bronzing of the skin or other manifestations to support it. I do not know just what is meant by this mass feeling more like kidney or glands than spleen. The dyspnea and drowsiness suggests some pulmonary or circulatory condition and we find later on that he has anemia enough to produce this although again we would like to know more about his chest. We find the note that he was unable to bear weight on his legs. We take it that this is because of weakness which is probably accounted for by his general condition and anemia. However this may be suggestive of something affecting the motor areas or the fibers which of course would have to be rather diffuse as there is no mention of there being any difference on the two sides.

It seems to me that the blood pressure in this case was rather high for a patient in this condition, but I suppose that this is nothing apart from the ordinary.

The reflexes as reported are rather indefinite and do not indicate anything tangible. The spinal fluid is negative so we do not have enough data to make a diagnosis of meningitis. However the vomiting and drowsiness, the motor paralysis of the hands which cleared up a little, points to something in the spinal cord. This could not very well be a localized involvement in the cord and that alone, however, as in such a case we should have exaggerated reflexes and Babinski. However, the symptoms point to some involvement in the sensations of needles and pins and the disturbance of touch sensation. It seems to me that even in presence of a negative spinal fluid and indefinite reflexes, we cannot rule out an involvement of the brain and cord.

Therefore, I shall venture a diagnosis as follows:

Based on data given: Epididymitis. Tuberculous and genito-urinary tuberculosis. Anemia secondary.

Speculative diagnosis: Meningitis, localized or limited, with possible tuberculoma.

DR. RICHARD C. CABOT

(In part from journal above.)

DIFFERENTIAL DIAGNOSIS

The background of the case so far as the previous history gives us any evidence is one of tuberculosis of the genito-urinary tract. I do not see that we have anything else that we are at all sure of. He has this lesion on the penis, the nature of which I do not know. I do not believe it is syphilis. Then after this long interval of fair health he comes in bleeding from the nose and mouth, with a moderate secodnary anemia, with a rather high blood pressure, and with pus in one urine specimen and not in another.

He certainly has not anemia enough to die from, but he perfectly well might die if another hemorrhage such as he has had happened to hit the nervous system, perhaps in the region of the medulla or perhaps in the ventricles.

If we take the account of the lumbar fluid literally, as I suppose we must, we must say that he had no meningitis. Anybody who is semistuporous and has a previous history of tuberculosis lesions makes us think first of tuberculous meningitis. But I think that can be ruled out here.

A paralysis coming on as rapidly as this cannot possibly, that I can see, be a cord paralysis in the absence of any demonstrable spinal lesion. Besides which his spinal fluid ought not to have been negative if he had something bulging into the cord from the vertebrae. I think we have to say that it is a brain lesion, and a brain lesion coming on more rapidly than any brain lesion that I know except cerebral hemorrhage.

That of course is only the terminal event. He had a secondary anemia and some bleeding before that. What the cause of that bleeding was I do not know. We have no study of the blood in relation to the different types of purpura. One cannot tell whether the platelets were decreased or not. It may be a thrombopenic purpura. That would go with cerebral hemorrhage as the terminal event. Tuberculosis seems to be one of the diseases which predisposes to thrombopenic purpura.

I do not see that we have any good reason to suspect nephritis, though since his blood pressure was a little high and his urinary gravity a little low, it goes through our minds as a possibility. I suppose the majority of cases of genito-urinary tuberculosis, we have reason to believe, in one kidney. He may have some in the other now. But without an examination of the urinary sediment and catheterization of the ureter I do not see how we can say more on that.

A thing that influences me against glioma is the knowledge that he was bleeding from other points, so that it seems quite possible that he might be bleeding in his brain. I do not think I can go any further on that. I think he has a focus of tuberculosis still in his genito-urinary tract. I think he has a purpuric type of bleeding, I suppose with low platelets, perhaps based on that tuberculosis. I think he has a cerebral hemorrhage. I do not mean to try to say where that cerebral hemorrhage is. My luck in predicting the localization of cerebral lesions has seldom been good.

DR. CABOT'S DIAGNOSIS

Cerebral hemorrhage.

Genito-urinary tuberculossi, prboably in the left kidney.

Purpura hemorrhagica.

DR. TRACY B. MALLORY

Anatomic Diagnosis: Tuberculosis of the left kidney.

Tuberculous ureteritis and cystitis miliary tuberculosis.

The case from beginning to end is one of tuberculosis. The brain on examination proved entirely negative, also the meninges. The lungs, liver, spleen showed miliary tubercles. The left kidney was considerably enlarged and was almost completely destroyed by caseous necrosis. The process had extended down the ureters. There were miliary tubercles the entire length of the ureters. There was tuberculous cystitis and prostatitis. The mass in the left upper quadrant may have been either spleen or kidney so far as we could determine. Both were enlarged, the spleen weighing nearly 300 grams, due to the miliary tuberculosis. The kidney was enlarged, probably both by the tuberculous process and as a compensatory hyperplasia for the absent kidney on the other side.

As to the respiratory paralysis I have only a suggestion to offer. On talking it over with the house officers afterwards it seemed to me that considerably more stress was laid on that than was perhaps justified. The duration of this symptom from its onset to death was only a little over an hour, in an extremely weak man who had just been given morphine. And when one comes down to it, all cases before they come to us do show respiratory paralysis.

ABSCESS OF THE LIVER AND ATYPICAL AMEBIC DYSENTERY (CASE REPORT)

R. B. RANEY, M. D.

(Discussed at the Staff Meeting of the St. Joseph's Hospital, Phoenix, Feb. 4, 1930.)

The following case, I think, will represent a group which go on to autopsy for an accurate diagnosis when they should be well understood in life with the proper interpretation of symptoms and the methods of diagnosis followed out in detail. I do not mean to say that a diagnosis should be made in all cases because that, as we all know, is beyond the possibilities of human endeavor, but there are a great many cases that are not thoroughly understood, not for a lack of knowledge regarding the symptom complex, but for failure of thorough study of the case at hand.

The patient entered St. Joseph's Hospital Nov. 1, 1929, and came under my observation Nov. 26 in consultation. The history obtained at this time was that of a progressive downward course, the initial onset in June of 1926. Chief complaints at that time were dyspnea, loss of strength, appetite and weight, with occasional passage of blood in the stool. This condition continued until February of 1929, when pain in the upper left quadrant developed. The pain was dull and aching in character as a rule, but occasional sharp seizures intervened. They were in no way affected by food or alkali. Confined to the area of origin without reference, and affected principally by change of posture. Patient noted that pain was more pronounced on standing than when lying down. However, this feature was only periodic and by no means constant. In summing up the points in the history from the onset to the time that this patient first came under my observation, there seems to have been a progressive downward course with a loss in weight from 150 to 100 pounds, dyspnea, loss of appetite and strength. Pain developed during the last six months of illness in the upper abdomen. Nausea on eating during last three months of illness, but no vomiting. However, eructation of mucus was quite pronounced during last three weeks. Passage of small quantities of blood at various intervals and otherwise apparently there was nothing of import-

(Continued on page 498)

CASE OF EPIGASTRIC PAIN AND CONTINUED FEVER

(No. 15141, Case Records of Mass. Genl. Hosp., from N. E. J. of Med., Apr. 4, 1929, p. 715.)

Case Record

An Irsih-American meat-packing checker forty-nine years old entered May 16 complaining of epigastric pain.

This pain began a year and a half before admission. At first it was a discomfort, "as if he were filled with concrete," and came on after and also before meals. Lately it had become sharp, darting through to the left shoulder, with pain over the heart followed by numbness of the left shoulder. It was now not related to meals except that he had a dull heavy feeling after eating heartily. When he took physic he felt a little better for a day or two. For six months his appetite had been getting very poor. He occasionally had some nausea, but did not vomit. His bowels had been irregular for a year and very constipated for six months. His stools may have been clay colored. He had never been jaundiced. After he passed the barium given him for x-ray examination he noticed a slightly bloody stool. His stools had always been very hard and he had always had to strain. He had rare night sweats. For six weeks he had had vertigo on stooping. Five weeks before admission he was obliged to give up work because he was tired and dyspneic. During the past three weeks he had lost much weight—about twenty pounds in the past two weeks—and had been very dyspneic on exertion.

His family history is not significant. He had been married twenty years. His wife had never been pregnant.

His past history is negative except that he formerly was "nervous" and did not sleep well. Recently he had slept better. Formerly he took a great deal of alcohol; for three months he had taken none.

At the Out-Patient Department May 2, two weeks before admission, it was further learned that food relieved his pain and that he had had much gas after meals and at night. He had morning headache. Examination showed slight tenderness in the epigastrium.

X-ray examination of the stomach showed no six-hour residue. The duodenal cap filled out normally. Peristalsis was normal. On the posterior wall of the fundus, particularly about the midportion of the stomach and the cardia, large filling defects were noted.

Clinical examination in the wards showed a pale man with dry skin and evidence of loss of weight. There was a small red area over the tenth vertebra. The heart was long and narrow (long thorax); no abnormalities except a soft systolic murmur at the apex. Pulses, arteries and blood pressure normal at admission. An electrocardiogram showed sino-auricular tachycardia, rate 140, diaphasic T. Liver dullness from the sixth rib to about two centimeters below the costal margin. Edge not definitely felt, but resistance was noted in the areas shown in the diagram. There was dullness in Traube's semilunar space and slight spasm in the left epigastrium. There was marked impulse in the right inguinal ring on cough. The fingers and toes were clubed. There was a red external polypoid tab protruding from the anus. No hemorrhoids were noted.

Urine: specific gravity 1.024 to 1.008, cloudy at six to thirteen examinations, alkaline at two, neutral at one. Benedict's test green at one, leukocytes at all of the last seven tests, sediment loaded at two, one red cell to great numbers of red cells at

(Continued on page 500)

CASE OF PONTILE HEMORRHAGE

B. M. BERGER, M. D.
Phoenix, Arizona

Discussed before the monthly Staff meeting of Good Samaritan Hospital, on Feb. 24, 1930.)

On June 29th, 1929, I was called to the home of this patient, a married woman, age 32. Her husband and two children, and father, mother, one brother and one sister, at that date were living and well. She went to work a 5 a. m., and returned at 8 of the same morning with no other complaint than feeling tired. This was shortly before becoming unconscious. No history of vomiting or gastro-intestinal disturbance. No headache. A hurried examination revealed the pupils fixed and contracted, breathing shallow with an inspiratory rather than expiratory effort. Rapid heart beat with evidence of enlargement on percussion, and no paralysis of muscles or limbs. She had clonic contractions of both arms and hands and rigidity of lower jaw. Systolic blood pressure was 65-70 and diastolic 0-5. Rectal temperature at least 111 F. checked by two graduate nurses and myself, twice within five minutes after entrance. Within ten minutes after entrance she became extremely cyanosed and expired. During the reading of death records for that month it was suggested that this was a case of heat prostration. I wrote the Journal A. M. A. asking for information and literature and the following is the reply

"This would seem to be a definite case of pontile hemorrhage. None of the cardinal signs are lacking. The history of a short interval of vertigo followed by a sudden loss of consciousness which is associated with contracted, fixed pupils and marked hyperpyrexia in a case which goes rapidly on to a fatal termination leaves little room for doubt as to the diagnosis. This history is unusually typical of a hemorrhage into the pons.

"As to differential diagnosis, it would seem that this case is unusually free from those elements on which differences of opinion can be based. Embolism of the medulla has been suggested; however, as there is no mention of any possible source of such emboli (endocarditis, thrombi, septic processes), or of any other embolic manifestations, this this diagnosis seems extremely unlikely. It is true that the information supplied does not explain the hemorrhage in a patient this young. Nevertheless such occurrences, though not common, are not a rarity and certainly are far more common than embolism without apparent source. Although there is no evidence pointing toward the presence of either syphilis or hypertension, they must be thought of as possible etiologic factors unless definite evidence against them can be presented. Academically at least a few other possibilities must be considered in differential diagnosis. However, these are readily ruled out. A cerebral hemorrhage, the usual type of apoplectic seizure, would be associated with dilated rather than constricted pupils and is not associated with such a marked hyperpyrexia, even terminally. A marked flaccidity of the extremities of one side or the other would most likely be present, and in addition a systolic blood pressure of 80 would be most unusual. Thrombosis of the vascular supply to either the medulla or the pons would be preceded by a much longer prodromal period which would undoubtedly be associated with definite signs of involvement of the medullopontile nuclei and probably of the cerebellum because of the intimate relationship of their arterial supply. A neoplasm involving the brain stem would also be much slower in development, and although the termination might come suddenly it would most certainly be preceded for several days or weeks by unmistakable signs. Acute bulbar palsy as defined by most authors includes both pontile and medullary hemorrhage. It also is referred to as acute inferior ploiencephalitis, which indicated an infectious state. Cases of an infectious nature exhibit no such rapidity of progression and termination as is exhibited here and usually are obviously of an infectious nature or associated with an infectious disease (usually in childhood). This rare condition has been thought to be closely related to acute anterior poliomyelitis so far as etiology is concerned.

As to differentiating between pontile hemorrhage and a hemorrhage into the medulla, it can only be said that the contracted pupils and marked pyrexia are typical of the former and certainly the pons was principally involved in this case. However, it would be impossible to say that the hemorrhagic process did not extend downward into the medulla and doubtless microscopic examination of these structures would indicate that it did."

Meager references to this subject are available in most neurologic textbooks.

In conclusion I wonder if, on account of the heat which seems mild and insignificant to old-timers of the Salt River Valley, we are not prone to ascribe symptoms to the pranks of temperature rather than definite pathology.

DISCUSSION BY DR. W. W. WATKINS

I believe the diagnosis of heat prostration, or insolation, is made too frequently on insufficient evidence. Dr. Berger became justly indignant when a nice, flossy diagnosis like pontine hemorrhage was clouded by comment from the Records Committee. If I were able to make a diagnosis of pontine hemorrhage from a patient's symptoms, I would feel aggrieved if some one were to come along and say, "O, it is just heat stroke." I offer for your edification, a brief summary of another case, full details and discussions of which will be in Southwestern Medicine when it comes off the press next week. This is not a local case, but one of Cabot's cases:

A Swedish carpenter, age 47, went to work on June 7th feeling perfectly well. It was a very hot day, being 94 in the shade in Boston; patient worked on the outside of the house and in the sun most of the time. About noon, he felt weak and dizzy and had to sit down; then vomited and was assisted home. Doctor called and found him in collapse, conscious but very weak, bathed in cold perspiration, mouth temp. was 94. B.P. 90/50, pulse weak and irregular but slow. He passed gradually into coma. The next morning his temp. was still subnormal: at nine o'clock temp. went to 104 and pulse to 150. At noon pulse was 160, resp. 16, B.P. 140/80. Patient died while being catheterized.

The interesting thing here is that all the clinicians who discussed this case diagnosed heat prostration, with which the clinical picture is consistent. Post-mortem examination showed the essential lesion to be hemorrhage and softening in the posterior third of the left cerebellar lobe; there was evidence of some increase of pressure under the tentorium, forcing the pons into the foramen magnum.

BOARD OF MEDICAL EXAMINERS OF ARIZONA

At the July, 1930, meeting of the Board, the following physicians and surgeons were licensed to practice medicine and surgery in Arizona:

JOSEPH D. VERTIN, Monrovia, Calif. Graduate of Loyola University, Chicago, 1916. By reciprocity with Illinois.

BENJAMIN HERZBERG, Phoenix, Ariz. Graduate of St. Louis University, 1929. By examination.

JAMES LEWIS JOHNSON, Chattanooga, Tenn. Graduate of Vanderbilt University, 1915. By reciprocity with Tennessee.

JOHN WILLIAM HUFFMAN, Florence, Ariz. Graduate of Northwestern University, 1930. By examination. Dr. Huffman will be associated with his father in practice.

CLARENCE E. BENSEMA, Tucson, Ariz. Graduate of University of Illinois, 1929. By examination.

JULES F. LaDURON, Superior, Ariz. Graduate of University of Louisville, 1920. By reciprocity with Indiana.

CHARLES N. SARLIN, Port Huron, N. Y. Graduate of Columbia University, 1921. By reciprocity with New York.

MYRO GILMORE WRIGHT, Denver, Colo. Graduate of University of Colorado, 1917. By reciprocity with Colorado.

At the October meeting of the Board, the following physicians and surgeons were licensed to practice in Arizona:

JOHN HUFFMAN WHITE, Rochester, Minn. Graduate of University of Oregon, 1926. By reciprocity with Minnesota. Dr. White will be associated with the Southwest Clinic in Phoenix.

HOBART B. STEWARD, Coolidge, Ariz. Graduate of St. Louis University, 1927. By reciprocity with Kansas.

HARVEY EDWARD MOSS, Kansas City, Mo. Graduate of Louisville Medical College, 1893. By reciprocity with Kansas.

JAMES J. LaSALLE, Phoenix, Ariz. Graduate of University of Michigan, 1896. Reciprocity with Ohio.

BLOOM BENSON, Tucson, Ariz. Graduate of Johns Hopkins University, 1926. By reciprocity with Illinois.

HAROLD HENRY RING. Tucson, Ariz. Graduate of Iowa State University, 1926. By reciprocity with Iowa.

EDW. HARRISON CALVERT, Scottsdale, Ariz. Graduate of Ohio State University, 1927. By reciprocity with Ohio.

MARCUS B. CIRLIN, Chicago, Ill. Graduate of University of Illinois, 1923. Reciprocity with Illinois.

ALAN C. SUTTON, Tucson, Ariz. Graduate of Johns Hopkins University, 1916. Reciprocity with Maryland.

HAROLD JAMES SCHILLING, New Hampton, Ia. Graduate of Marquette University, 1927. Reciprocity with Iowa.

PERCY BROWN, Boston, Mass. Graduate of Harvard University, 1900. By reciprocity with Massachusetts.

JOSEPH JAMES BOUCHER, Hartford, Conn. Graduate of College of Physicians and Surgeons, University of Maryland, 1904. By reciprocity with Connecticut.

VIVIAN TAPPAN, Tucson, Ariz. Graduate of Johns Hopkins University, 1927. By examination.

HEBER O. TUCKER, Phoenix, Ariz. Graduate of Johns Hopkins University, 1929. By examination.

JACK R. HILD, Douglas, Ariz. Graduate of Tulane University, 1929. By examination.

CARL EUGENE ETHERIDGE, Phoenix, Ariz. Graduate of University of Arkansas, 1929. By examination.

CLARENCE L. ROBBINS, Tucson, Ariz. Graduate of University of Arkansas, 1929. By examination.

DWIGHT M. DETER, Tucson, Ariz. Graduate of Baylor University, 1929. By examination.

COURSE IN ENDOCRINOLOGY IN PHOENIX

DR. E. KOST SHELTON, Santa Barbara, California, first assistant to Dr. William Englebach for the past several years, will be in Phoenix to give a course upon Endocrinology. There will be six two-hour lectures. The lectures will be illustrated and will embrace the following:

1. Introduction.
   Autonomic nervous system.
   Pituitary—Hypo-and hyper-activities of both the anterior and posterior lobes.
   Pituitary tumor.
2. Thyroid—Hypoactivities. Cretinism and myxedema.
3. Thyroid—Hyperactivities. Goiter (colloid, exophthalmic and adenomatous.) Pituitarothyroid and thyropituitary disorders.
4. Gonads—Hypo-and hyper-activities in both male and female.
5. Parathyroids—Hypo-and hyper-activities. The newer conceptions of calcium metabolism. The suprarenals (medulla and cortex).
6. The thymus and pineal—Pluriglandular disorders. Clinic.

Dr. Shelton has the following to say: "Each subject would be treated about as follows: A brief resume of the phylogenesis, embryology, anatomy, histo-pathology and physiology of the gland, its relationship to the entire endcorine system and other systems of the body, its clinical significance and modern treatment of its disorders. Of course this takes up a lot of modern biochemistry, particularly regarding the anterior lobe of the pituitary and the gonads. The lectures would be entirely practical from a clinical standpoint, however, and be or interest and value to every man no matter what his specialty."

The fee for the course is $25.00 providing there are as many as ten men taking the course.

Those who wish to take the course will please apply to Dr. Orville Harry Brown.

DALLAS SOUTHERN CLINICAL SOCIETY

Elsewhere in this issue will be found advertisement of the second annual Clinical Conference of the Dallas Southern Clinical Society, to be held from March 30th to April 3rd, 1931. This is very advantageously plcaed immediately following the Congress of the College of Physicians in Baltimore which will close on March 27th.

The Dallas meeting was a very successful one this year (1930), and a very attractive program is in the process of construction for the 1931 Conference.

SITUATIONS WANTED

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MEDICAL & SURGICAL ASSOCIATION OF THE SOUTHWEST

DR. E. PAYNE PALMER ..... Phoenix
DR. W. WARNER WATKINS ..... Phoenix

WHAT TO DO IF THREATENED WITH SUIT FOR ALLEGED MALPRACTICE

(The following outline is furnished by Dr. D. F. Harbridge, Secretary of the Arizona State Medical Association, and applies particularly to the physicians and surgeons of that state.)

A physician threatened with a suit for alleged malpractice usually receives his first intimation of the same in the form of a letter from an attorney, stating that his former patient has recently sought his legal advice, and that considerable trouble might be saved by all concerned by an early interview between the physician and the attorney.

What should a member of the Arizona State Medical Association do under such circumstances?

He should, without comment, promptly acknowledge receipt of the communication, and state that further reply would no doubt be made in due time by his (the physician's) legal representative (not necessary to name.) The threatened member should **immediately** send copies of this correspondence to the Secretary of the Arizona State Medical Association. This done, **there is no occasion for haste**, and no occasion for actually consulting an attorney until after a formal summons from the Superior Court has been served by a representative of the court. Such a summons always mentions a time limit, usually twenty-one days, during which an appearance must be made in court in behalf of the physician upon whom summons has been served. This period leaves ample time for the completion of a formal application by the member for the assistance of his State Medical Association in the conduct of his defense.

Every step in the requirements printed on the application blank is based on fifteen years' experience in the administration of the medical defense benefits of our State Association, and is designed to acquaint the Medical Defense Committee, and the legal counselor for the Association, with the charges against the threatened member, as well as his recital of his methods pursued in treating the case. The names of consultants called, if any, and the office or laboratory and hospital records of the case available for developing plans for the conduct of the defense in court are thus reviewed by certain of the threatened physician's fellow-members. As a result of this care in approving applications for defense by the State Association, many threatened suits in the past have been abandoned and those coming to trial have always heretofore been successfully defended.

In conclusion, all members are advised to pay their annual county medical society dues promptly; second, to keep records of all patients; third, to seek consultation in unusually difficult cases; fourth, to require patients refusing x-ray examinations, when requested, to sign a release blank; fifth, tolerance and exercise of the severest caution not to criticise before a patient the work of a colleague; and, finally, to consult promptly the secretary of the county medical society or the state association before discussing with an attorney any threat based on alleged malpractice.

SIXTH BIENNIAL REPORT OF THE NEW MEXICO BUREAU OF PUBLIC HEALTH.

This is a very handsome brochure, one of those we like to file away for future reference. Handsomely printed, on fine paper, it contains some very interesting data. It summarizes by graphs the information previously given in a paper by Dr. C. S. Luckett, Director of the Bureau. It then summarizes the activities and work of the Bureau during the two year period from July 1, 1928, to June 30, 1930.

ARIZONA STATE BOARD OF HEALTH MONTHLY BULLETIN

The August issue of the Bulletin of the Arizona State Board of Health is a very pleasing pamphlet. It is pleasing because it gives some very interesting information in readable type, and because it apparently intends to inaugurate a new series of monthly bulletins for that state. The article on the "Full Time Health Units for Arizona" is worthy of quotation in extenso:—

THREE FULL-TIME COUNTY HEALTH UNITS FOR ARIZONA

The greatest advance in health yet made in Arizona, according to U. S. Public Health Service Standards, and other health agencies interested in the promotion of the prevention of disease, has been the establishment of a full time unit in Pima County, the first county in Arizona to set up. This is due to the cooperation between the state board of health, the board of supervisors and the county board of health of Pima county.

The unit opens with a county director of public health; three nurses; a sanitary inspector and a clerk.

Gila county is the next, and has also signed for its full-time unit, providing for a county director of public health; a nurse and a sanitary inspector.

In this connection the cities of Miami and Globe have joined with the county in forming this unit, by appointing the same personnel as the board of supervisors. Thanks are due them for this cooperation.

The Board of Supervisors of Maricopa has just signed a budget of like intent, which provides for a full-time health unit in that county, with a health officer; two health nurses; two sanitary inspectors and a clerk, together with ample provision for laboratory purposes.

With these three counties fully organized, approximately 55% of the entire population of the state of Arizona is under full-time units—this with Cochise, Yuma and Coconino, which have full-time health and indigent officers, gives Arizona practically 43% of its counties under full-time men. Provision has been made by the U. S. Public Health Service for at least one more county to be set up.

For the period beginning July 1st, the State Superintendent of Public Health has been able to obtain from the U. S. Public Health Service and the Rockefeller Foundation, the total sum of $17,100 to apply on county health units. This amount of money for this next year is almost equal to the total amount appropriated to the state board of health, namely $21,400. Altogether, beginning January 1st, 1930, Arizona has been allotted funds from the U. S. Public Health Service and the Rockefeller Foundation, a total of $27,000, or more than one year's appropriation. The proper handling of these funds and the success of the county health units will predicate further help from these and other outside agencies. It is clearly up to the people of Arizona to get back of these health units to see that they function properly. These health units, in order to accomplish the best results, must be free from outside influence which would tend to throw away the public good for personal desires.

It is believed that here in Arizona the consciousness of the people has been awakened and that the intelligent part of our population, which is unusually high, will prevail in making Arizona the best state in the union from a public health viewponit.

SCIENCE AND CULTISM

The editor has been holding on his desk, for several months, the editorial which appeared, under the above title, in the Journal A. M. A. for Feb. 1, 1930, waiting until the memory of it has had time to grow dim in the minds of the readers of that journal, in order that we might reprint it with proper effect. There is meat here for thought and material for use of those who will discuss this matter with legislators during the coming months of trial, when the medical profession will engage in its ever recurrent fight to protect an unappreciative public from quacks and cults:—

"Now and then the medical profession is upbraided by the proponents of various notions in the field of health and science because it fails to give to their claims what they conceive to be adequate consideration. Again and again the difficulties of Galileo, Harvey, Jenner and Pasteur, when they attempted to convince the leaders of their times of the importance of their discoveries, are cited as evidence that scientists are intolerant. Apparently cultists and others who have had but little experience in reasoning and logic, or with what is known as the scientific method, fail to take into account the fact that the world has moved since the time of the prophets, and that science has advanced more in the past fifty years than in the previous fifty centuries. James Harvey Robinson wrote an interesting essay on "The Importance of Being Historically Minded." With a proper perspective, one realizes that science is today in a position to demand evidence to an extent that might not have been warranted in a previous period when the whole world was dominated by magic and mysticism.

Recently, Mr. Chester Rowell, feature writer for the San Francisco Chronicle, discussed the appeal for tolerance made by faith-healing cults in the Los Angeles Times, following an expose by the editor of The Journal of some of the weird quackeries existing in Los Angeles. Mr. Rowell says:

But the appeal for "tolerance" by one "school" or another, is an example of a common fallacy. There is no "tolerance" of astrology by astronomers. There is no "tolerance" of fortune-telling by psychologists, nor of perpetual motion inventors by physicists. Geologists do not locate oil or water by dowsing with a forked stick, nor "tolerate" those who do. Entomologists do not "tolerate" those who would exterminate insect pests by interfering with their spontaneous generation. Scientific agriculture does not "tolerate" the theory that potatoes grow wrong unless planted in the dark of the moon. All these "schools" exist, and they are all rejected outright as unscientific superstitions by every scientist in the world.

On the other hand, good Catholics tolerate the Holy Rollers and Buddhists tolerate the Mormons.

Atheists tolerate the faith of Christians and Christians the unfaith of atheists. Protestants and Christian scientists tolerate each other's religion, each respecting the right of the other to seek God in his own way. But the law of the land did not tolerate polygamy, when the Mormons said it was religion, and the Regents of the University of California do not permit the anti-vaccinationist student to endanger the health of other students, even though he calls his objection religious.

So in medicine. If it were a matter of faith, dogma or canons, one "school" should "tolerate" anothre. If it is a matter of science, then the only distinction is that of scientific and unscientific. And between science and non-science there is no equality of right, and no basis for tolerance. The fact that millions of devout people in India believe in casting their horoscopes by the stars does not erect them into a "school" of astronomy, nor impose on astronomy any obligation to recognize them. They are neither "regular" nor "irregular" astronomers—they are not astronomers at all. Neither is any unscientific theory or practice of healing any part of the science of medicine. There are only two sorts of medicine, scientific and unscientific. And of the unscientific "schools," science has only this to say—that they are unscientific.

How, then, shall we distinguish which principles and practices of healing are scientific, and which are not? The simplest test is that which we unhesitatingly apply in every other branch of knowledge. That is the judgment of scientists. If the scientists say that a certain thnig is scientific, we accept it as such. If they all say it is unscientific, we say likewise, at least until it has succeeded in convincing them. Every scientific university in the world teaches astronomy, and not one teaches astrology. All of them teach chemistry and not one teaches alchemy. Every university in the world teaches scientific medicine, and not one of them—not a single one in the whole world—teaches or recognizes any of the "schools" or sects for which the Times speaks. If the unanimous voice of science means anything, this is its verdict.

The next test, and the decisive one, is that of method. Scientists may be mistaken, sometimes, in their results and conclusions. Sometimes a thing which seems true in the light of incomplete information becomes only partly true in the light of later discoveries. But science is not mistaken in its method. That method is systemtaic observation and experiment, and the submission of these observations and experiments to the scientists of the world, for them to repeat, to test and to scrutinize. Whatever pursues that method and is approved by that test is scientific—including, in medicine, light rays for tuberculosis, diet for many ailments and hydrotherapy for certain mental conditions. Whatever does not proceed by that method, or fails by that test, is unscientific—including all the cults, sects and schools which Dr. Fishbein rejects and the Times defends.

Mr. Rowell has placed his finger unerringly on the weaknesses of the cultists. His logic might well serve as a text in the schools, not only that it might aid the younger generation in learning the art of reasoning and judgment, but also that physicians might realize the basic folly of the strange schemes which are constantly being introduced to the public around them. —Jour. A. M. A., Feb. 1, 1930.

ABSCESS OF THE LIVER AND ATYPICAL AMEBIC DYSENTERY

(Continued from page 493)

ance in the history. No history of diarrhea. In fact, patient specifically stated that he had never had any trouble with diarrhea, neither had he been afflicted with constipation.

Physical examination at this time disclosed a white male, approximately 50 years of age, with a marked degree of emaciation. On first appearance one would be immediately struck with the typically cachectic state of advanced carcinomatosis; however, on second thought there was something that suggested sepsis.

The abdominal examination disclosed a large mass in the upper abdomen which could be seen by inspection, non-pulsating in character and certainly not nodular. It was soft and boggy in consistency, without definite outlines, pulsating slightly on pressure and non-movable. There was a distinct murmur over this area, fading out upward towards the precordial region and downward over the abdominal aorta, synchronous with the cardiac impulse. There was little or no rigidity over the mass and only a moderate amount of tenderness. Rectal examination disclosed considerable tenderness and an edematous area over the prostate gland, proctoscopic examination being advised. There were no hemorrhoids, fissures or evidences of new growth. Aside from these findings the examination was negative, except of course for the advanced degree of emaciation.

X-ray examinations that were noted are as follows: November 1st, 1929, there was apparently a single plate taken in the region of the spleen with the report that there were no abnormal densities in that region or elsewhere in the abdomen. Subsequent to this report there was an examination of the stomach which showed contour of the pylorus normal, the duodenal cap showed no filling defect, nor was there other evidence of pathology. This was followed by a colonic examination. The barium snema would not pass beyond the iliac colon. There was no gross filling defect in this area. The barium given the previous day passed out of the colon and the impression was that this was a spasm of the bowel rather than an organic obstruction. Urine showed a heavy trace of albumin. R. B.C. 5,000,000; hemo, 70; leukocytes 14,900, polys 89. Gastric-analysis of Nov. 11 showed an acidity of 21, free hydrochloric acid 10. No occult blood. Wassermann examination was negative.

From Nov. 1 to Nov. 27, the course was progressively downward, afebrile in character with the exception of an occasional rise to 99 degrees intermittently. Treatment was diet with rest in bed. Occasional hypodermic of morphine was given for pain. In summing up the cardinal features of this condition, the following points seem most prominent. General downward progress, onset of pain of the character and tpye described, blood in the stool, rectal tenderness, evidence of tumor mass in the upper abdomen with its pulsating characteristics, the elicitation of a murmur confined to the mass, synchronous with the cardiac impulse, which died out downward over the abdominal aorta. The absence of positive findings on x-ray of the gastro-intestinal tract. The diagnosis noted on the hospital chart was cancer of the pancreas.

In summing up the salient features I can hardly agree that this patient is suffering from a malignancy of any type but would be rather inclined to think that in all probability there is a generalized infection and even in the face of a negative Wassermann, I would still suspect the general condition to be luetic in origin. The mass in the up-

per abdomen, I think presents enough characteristics that it might be called an aneurism of the abdominal aorta. However, I am putting forth opinion in the form of a working diagnosis for want of something better, and am not inclined to think that this patient's condition can be bettered in any way surgically. However, the object of consultation was to determine whether or not an exploratory laparotomy should be advisable and this procedure I think should be left to the discretion of the patient, which he decided on and requested the following day.

Operation: Exploratory laparotomy, Nov, 27. Under novocain anesthesia, midline incision made starting high up on the ensiform and extending downward to the right and little below the umbilicus. Liver and gall bladder were normal in appearance and position. The stomach was examined and appeared rather whitish in color probably due to the patient's condition but no masses, ulceration or adhesions were found. There was marked retroperitoneal edema but no definite mass could be made out. There was a large pulsating aorta but could not be interpreted as an aneurism. Intestine was pale but probably normal. In the region of the cecum there was boggy like mass infiltrating the wall, soft and irregular in shape, extending up about half way on the ascending colon and involving the entire corcumference of the cecum. There was marked venous congestion in this region. Cecum was mobilized with difficulty. Appendix was retrocecal but normal in appearance. The ascending colon was found normal including the hepatic flexure to an area about half way across transverse colon. Another soft boggy nodular mass involving wall of colon was felt with slight ulceration on the peritoneal surface of the colon where another similar but smaller condition was found. The sigmoid could not be satisfactorily examined. Neither kidney could be satisfactorily examined because of the retroperitoneal edema.

A section was removed from the cecum and sent to the laboratory for pathological examination.

Abdomen was closed in the usual manner. Patient was given 500 c.c. of saline solution in the vein.

COMMENT: It is my impression that this pathology in the colon is probably secondary to pathology elsewhere. There is still possibility of some retroperitoneal tumor but because of the edema, this could not be made out.

DIAGNOSIS: Undetermined.

Patient expired Dec. 2, after a rather stormy post-operative course.

AUTOPSY: Examination was negative with the exception of the colon and the liver. There was a large abscess in the left lobe of the liver containtaining approximately four ounces of pus, which broke through at the diaphragmatic surface. The surrounding edema in the soft tissues explains the murmur heard in this region, and also the mass described in the physical examination. On opening the colon, there were multiple ulcerations along the entire surface extending from the cecum to the sigmoid, characteristic of amebic infection. The abdomen was filled with plastic exudate due to perforation of numerous ulcerated areas and resulting peritonitis which was the direct cause of the patient's death.

DISCUSSION

Dr. Joseph Madison Greer

In the discussion of a case of this kind it is probably in order to say a few words about amebic dysentery in general.

Amebic dysentery has been defined as an acute infectious disease caused by entameba histolytica and characterized by the presence of multiple ulcers in the colon, which show a tendency to coalesce and to produce communicating sinuses in the submucous tissue and amebic abscess of the liver.

The exciting cause is of course the entameba histolytica, and as to the predisposing factors we usually think it is necessary for a patient to have been in the tropics in order to become infected with ameba. This case shows that this is not at all necessary as he was never in the tropics. Dr. Dysart has also called our attention to this point and has made the statement that he has seen cases of amebic dysentery in this section of the country that have never been in the tropics.

The adult male seems to be more often infected than either women or children. This is probably because of the water supply. The incubation period is given from seven to twelve days. The disease develops insidiously with a mild diarrhea for a while and then constipation. Then alternate attacks of diarrhea and constipation. There is gradual loss of strength and in advanced cases the patient may be unable to leave his bed. There are frequently complaints of ringing in the ears, anorexia and occasional attacks of nausea. There may be some fever at first but later the temperature falls to normal or below. Upon physical examination we usually find evidence of emaciation and associated signs, such as yellow skin, sunken cheeks, dry tongue, etc. There is also flabby muscles, edema of the ankles and later weak and rapid pulse.

During the attacks of diarrhea there is the passage of bloody mucus and amebae should be found. However in the chronic cases it is not always easy to find amebae. The blood findings should be that of anemia.

Of the complications of amebic dysentery, hepatic abscess stands out as the most important, although there are other complications as peritonitis, pyemia, malaria, bronchopneumonia, etc. Hepatic abscess may develop at any stage of the disease but usually after the disease has become chronic. The abscess is usually located in the right lobe near the superior surface, the organ as a rule is somewhat enlarged. In this case there was no appreciable enlargement of the liver and the abscess occupied the left lobe of the liver near the superior surface and replaced the liver substance rather than displacing it.

It would seem in this case that a diagnosis should have been made. If this man had a history of diarrhea, or if he had ever been in the tropics, the first sight of the colon wolud have caused an immediate exclamation of "amebic dysentery." If he had been a soldier or if I had operated upon him in an army hospital I am quite sure that I should not have been led astray. However, I am frank to say that I was so sure that there was a malignancy someplace that I did not think of a liver abscess even after exploring the abdomen and inspecting the colon. This was a case in which the history was misleading. Usually the history is the most important help in the diagnosis.

DOCTORS AND SPECIALISTS, by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association. Bobbs-Merrill Co., 188 pp. $1.00.

The subtitle of this brochure is "A Medical Revue with a Prologue and a Good Many Scenes." It is dedicated to "Every Doctor with a Sense of Humor, —Something a Doctor Must Have to Live at All." This is Fishbein's gentle, at times pointed satire at its best. Every doctor will enjoy this book. If he is a specialist, he will enjoy the caricature of himself which he will likely find there. After buying and reading it, let your friends read it and laugh with you. W. W. W.

CASE OF EPIGASTRIC PAIN AND CONTINUED FEVER

(Continued from page 493)

six of the last eight. Blood 11,100 to 28,400 leukocytes, 67 per cent polymorphonuclears, hemoglobin 60 to 70 per cent, reds 4,020,000 to 4,840,000, moderate achromia. Two Hinton tests strongly positive, one Wassermann moderately positive. Fasting contents of stomach: 15 cubic centimeters of thick clear mucoid fluid, no free hydrochloric acid, combined acid 2, guaiac negative. Test meal: thick yellow mucoid fluid, no free hydrochloric acid, combined acid 13, guaiac strongly positive. Stools, guaiac positive at ten of thirteen tests, strongly to very strongly at eight.

X-ray examination showed the diaphragm low on the right, high on the left. The costophrenic angles were clear. There was generalized prominence of the larger lung markings. The aorta was slightly tortuous. The kidney outlines were rather indistinct on both sides. There were two shadows on the right, probably artefacts. The spine showed an anatomical variation in the fifth lumbar vertebra. The plate was unsatisfactory on account of motion. Plates taken with the radiographic catheter in position confirmed the previous findings. After injection, the pelvis of the right kidney was fairly well shown lying unusually low, opposite the transverse process of the third (?) lumbar vertebra. The lower pole of this kidney overlay the crest of the ilium. The pelvis was not enlarged or abnormal in shape. The usual cupping of the minor calices was present.

Temperature 97.7° to 102.2°, with elevation to 104.8° May 20 and a terminal rise to 105°. Pulse 75 to 131. Respirations not remarkable.

A surgical consultant advised exploration. One of the surgical chiefs advised against it.

The morning of May 20 there was a rise of temperature to 104.8°. The leukocyte count was 28,000. There was marked costovertebral tenderness on the right. The patient vomited but complained of no pain. That night the abdomen was slightly tense. The next day he vomited again. There were a few small blood clots in the vomitus. May 23 the temperature was nearly normal. In the right costovertebral angle a small firm irregularity was felt, quite tender to pressure. A genito-urinary consultant thought the shadow in the region of the right kidney might be a stone. A visiting physician believed exploration was warranted. The patient felt well. June 1 cystoscopy showed both ureters negative. The shadow did not show in the plates taken at cystoscopy. No evidence of renal pathology was found.

June 5 a blood culture showed streptococcus viridans. The organism differed from those isolated from subacute bacterial endocarditis cases both in morphology and cultural character. It was closely allied to organisms found in a few cases of arthritis and some rheumatic aortic heart valves at autopsy. June 9 another blood culture was negative.

June 11 a genito-urinary consultant thought there was a mild cystitis following cystoscopy and thought it was not a factor in the elevation of temperature.

The patient grew increasingly drowsy. He had almost constant hiccup for three days before death. June 18 he died.

DISCUSSION

DR. FRANK J. MILLOY

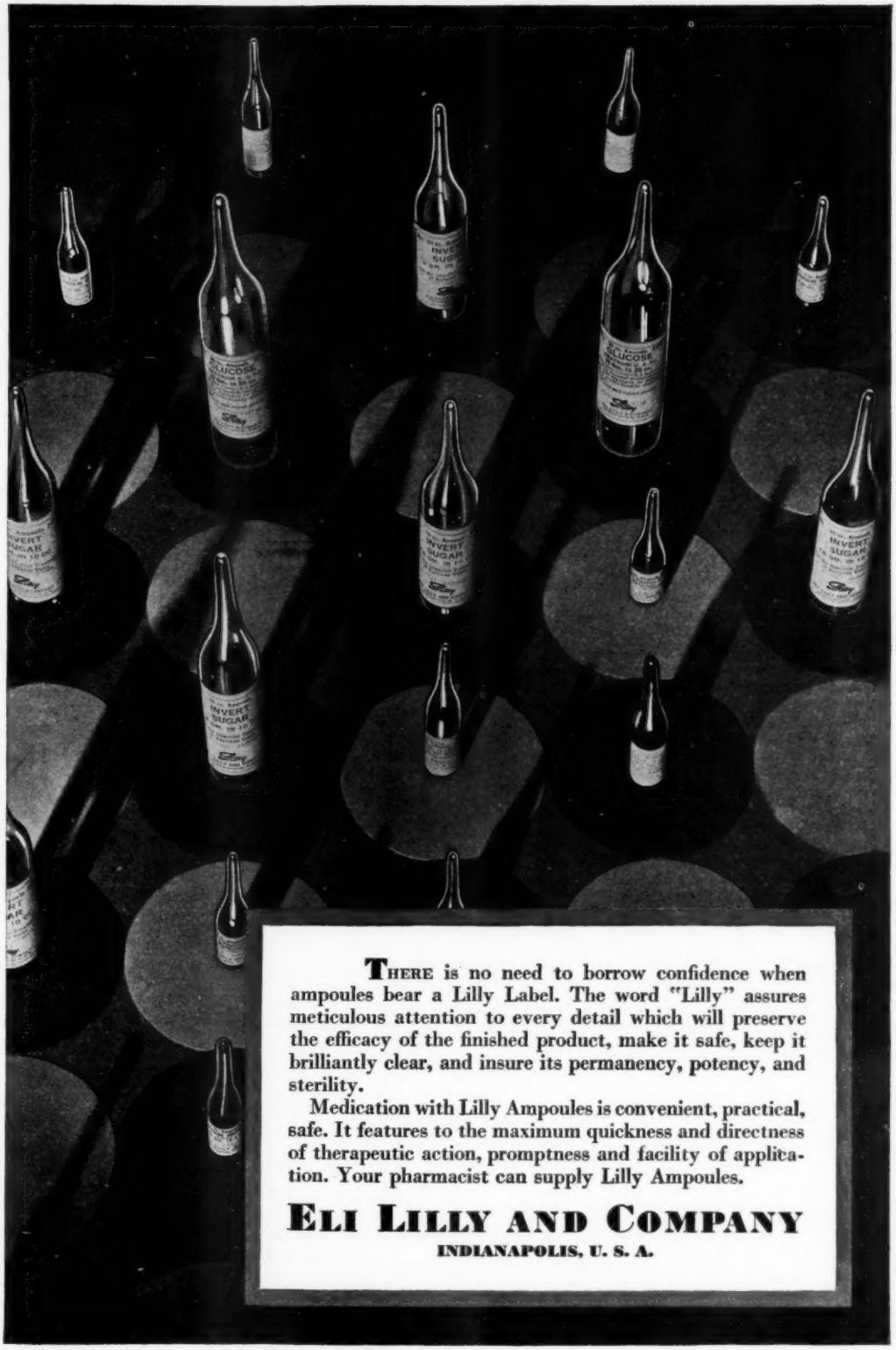
(Before Clinical Club of Phoenix)

At first thought this looks like a complication of diseases. But I think it will resolve itself into that of a man with an old luetic infection who has developed carcinoma of the stomach. The main point of differentiation is between carcinoma and syphilis of the stomach, and while syphilis may simulate any other disease, this is almost too typical a picture of carcinoma of the fundus to be syphilis. Carcinoma will explain any symptom here except the urinary findings and positive blood culture obtained at one time.

The age of onset (at 49), the history of onset beginning a year and a half before admission, the heavy feeling in the stomach at the onset, and the discomfort relieved at times by food, the feeling of relief after a physic, the gradual loss of appetite, the loss of weight and strength, and the stubborn constipation during the past six months, present a typical picture of carcinoma. The x-ray localizes the lesion in the fundus. This explains the slight nausea and absence of vomiting and other evidences of obstruction, because there was no organic process in the region of the pylorus. The stomach analysis is a little misleading; the total acidity of 13 and the absence of free HCl is more like gastric achylia. If a test for rennet had been made it would have decided this point. Rennet is absent in gastric achylia. In carcinoma you expect an absence of free HCl but a high total acidity. However, gastric achylia is a common finding in old luetic cases, without any other evidence of syphilitic involvement of the stomach, and this man may have had a gastric achylia before ever developing a cancer. There was no test made for lactic acid and nothing said about Boas-Oppler bacilli, but would not expect to find these present when there was no obstruction. The strongly positive guaiac test in the stools is one of the most important findings in favor of carcinoma of the lumen of the gastro-intestinal tract. There are only about seventy cases of gastric syphilis reported in the literature. It occurs in three forms; as an ulcer in the region of the pylorus simulating ordinary non-specific peptic ulcer, as an extensive infiltrating process in the wall of the stomach, or as a gumma. It seldom gives the strongly positive guaiac reactions on the gastric contents and stools, and very often there is a barium residue in the stomach. There was no residue in this case and if the observation had been made, it would probably have been found that the barium left the stomach rapidly, which is further evidence of carcinoma not involving the pylorus. But the main evidence against gastric syphilis is the fact that he did not respond to anti-luetic treatment, or else did not receive any.

Of course it is impossible to tell just how many of these terminal pains and other symptoms may have been caused by syphilis and how many by the carcinoma. Syphilis may explain the electrocardiographic findings. I think they are trying to find evidence of a hypernephroma aslo. X-raying the spine and searching for tumor in the region of the kidneys suggest this, but I cannot see where they have found any evidence. Hypernephroma is an affection of childhood or advanced age. Hematuria is almost a constant symptom. The normal pyelograms are against hypernephroma or other kidney tumor. There were a few blood cells at first but the blood cells in the later specimens were probably the result of the cystoscope. But this man may have had renal calculus and pyelitis. There is sufficient proof for this and hardly anything else will explain the blood and pus in the urine.

I do not know how to explain the positive blood culture. I doubt if it will be explained. There is no other evidence of endocarditis nor sepsis, and there is no evidence of nephritis. The variation in specific gravity, absence of albumin and edema, and normal blood pressure and arteries, are against any nephritic disease. The elevation of temperature and leucocytosis are not uncommon in carcinoma. The terminal hiccough may have been due to the



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carcinomatous process in the upper abdomen, or to interference with renal function toward the end.

Diagnosis:—Syphilis nad gastric carcinoma.

Diagnostic conclusions on this case were given by the following members of the Club:

Dr. F. C. Jordan:—Syphilis; carcinoma of stomach.

Dr. H. P. Mills:—Carcinoma of stomach; endocarditis, mitral and aortic; pyelitis of right kidney.

Dr. W. W. Watkins:—Possibilities from history, heart or aortic disease, stomach lesion, gastric crises, liver or gallbladder lesions, pancreatic lesions, abdominal angina, colon lesion. Clinical examination rules out heart; laboratory and x-ray establish stomach lesion, probably cancer and syphilis. Diagnosis: cancer of stomach, posterior wall with perforation, and septicemia.

Dr. T. T. Clohessy:—Syphilitic aortitis; gastric syphilis on which developed a streptococcic viridans septicemia. Gastric symptoms, achlorhydria, lung markings quite characteristic of syphilis; final illness and death from streptococcus viridans septicemia.

Dr. O. H. Brown:—Streptococcic endocarditis (mitral); cancer of stomach; syphilis; pyelitis and ptosis of right kidney; aortitis; hernia.

DR. RICHARD C. CABOT

(In part, from journal above)

We certainly cannot conclude much from that epigastric pain. It is about the commonest place for pain in the human anatomy.

The first two sentences in the present illness suggest angina pectoris. But the pain may be due to distention of the stomach with air.

Among the many symptoms noticed the dyspnea is perhaps the most outstanding. We wonder if he has a cardiac trouble or a hypertension. We wonder if he has a nephritis which has gradually gone on to produce his anemia. We wonder about his stomach on account of the gastric symptoms. We wonder about his gall bladder, but not very definitely. That is as much as we can do on the basis of that first long paragraph.

"It was further learned that food relieved the pain and that he had had much gas after meals and at night." That means they thought of ulcer.

X-ray examination of the stomach is very important in a case like this. Dr. Holmes, will you be good enough to comment on these plates for us?

Dr. George W. Holmes I might say first that the gastric examinations at this hospital are made largely with the fluoroscope. We depend very little on the plates in the final interpretation of the x-ray findings. When we find anything interesting by fluoroscope we take plates to demonstrate it. This patient presents a deformity at the fundus of the stomach. From these plates you would not be able to say whether there was anything wrong with the pyloric portion or not. They were taken to demonstrate the deformity in the fundus of the stomach. Deformities in this region are the most difficult to demonstrate of all lesions of the gastrointestinal tract. Lesions of considerable size may be completely missed in this region. Success in demonstrating it depends upon watching the barium as it passes into the stomach and on examining the patient in a great many positions. The observation which is most valuable is the passage of the first swallow into the stomach. Another observation that is valuable is made with the patient on his back on the couch. When the patient breathes deeply the normal stomach changes in shape with each expiratory excursion. In a stomach infiltrated with cancer this change does not take place. In the lower portion of the stomach our diagnosis is much easier because we have peristaltic waves which we can see passing over the stomach. This patient shows a definite deformity, and we have the other signs, so that we have enough evidence to justify us in making a diagnosis of tumor of the stomach. A lesion at the fundus is almost invariably carcinoma. You do not get ulcer in that region. There are very few benign lesions to consider. In fact I cannot recall a single lesion in that region that was not cancer.

Dr. Cabot: Working with one or another radiologist one gets used to the different temperaments. Dr. Holmes is a particularly conservative man. In all the years we have been working together never

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have I heard him commit himself so emphatically. I cannot help but be strongly moved.

"Liver edge not definitely felt, but resistance was noted in the areas shown in the diagram." Of course with a diagnosis which we suspect strongly of being carcinoma of the stomach we are thinking of metastases in the liver.

The dyspnea on exertion is interesting, because we are so apt to associate that with heart and lung disease, although we see many cases here where it is not associated with any disease of the thorax, many cases with high diaphragm or tumor below the diaphragm, raising it, and a good many cases which we cannot explain at all.

NOTES ON THE PHYSICAL EXAMINATION

I do not yet know why his fingers were clubbed.

There is nothing extraordinary about the urine examination. We cannot construct any particular lesion in the kidney out of that.

The blood shows a slight secondary anemia with leukocytosis.

From the x-ray examination that could not be syphilis of the stomach, could it?

Dr. Holmes: I do not believe so.

Dr. Cabot: So that what you would expect to see if it were a cancer is present in the stomach examination. Excluding rare meat diet and hemorrhoids that guaiac is suggestive of an oozing carcinoma higher up.

Dr. Holmes: This is the left chest. The diaphragm is distinctly higher on the left. There are a number of things that can give high diaphragm on that side, among them carcinoma of the fundus of the stomach and tumor immediately below the diaphragm. But high diaphragm is very common when there is nothing wrong. A patient who has just eaten will have a high diaphragm on the left side. Anything that interferes with the passage of air into the lung will cause a high diaphragm. There is nothing in this patient's chest, nothing in the lung or bronchus to suggest such a condition. Our interpretation would be that he had something below the diaphragm pushing it up or that he had taken food just before the plate was made.

In the x-ray of the kidney there is a suggestion of a shadow in this area. There was some question at that time as to whether that was a stone in the lower pole of the kidney or whether it was an artefact. By artefacts we mean shadows produced by substances on the skin or due to some error in the dark room. It is hard to say, but I think that is an artefact. There is something about it that suggests it.

Then he had a catheter inserted. The right kidney is considerably lower than the left, I think perhaps a little more than normally, and the shadow whoch we saw in the previous plates is not visible in this, at least in this light. So that that would rule it out as being a stone and place it definitely in the category of artefact.

Dr. Cabot: So we find nothing to trace to the kidneys. In connection with the dysuria remember the leukocytes and blood which we noticed in the urinary sediment. In spite of many complaints which we do not satisfactorily account for, and although there is some blood and pus in the urine, we do not seem to have any sufficient evidence to condemn the kidney.

Dr. Edward L. Young, Jr.: He had, following this, temperature and costovertebral tenderness. I think it is true that pyelitis is often made to take the blame when temperature is not otherwise explained, and I think there is often unwarranted curiosity on the part of all of us to find out the why and wherefore. It seems to me that even if that was a stone we took the chance of stirring up the kidneys by cystoscopy.

From May 20 to the fourth of June is a very long time for it to take a case of infection in the kidney to develop. In a coccus infection you will get the cocci in the urine, but you may get a grossly clear urine. But even May 20 to June 4 is a long time for that to stay clear. I should question somewhat whether there had been some pyelitis present.

Dr. Cabot I will read to the end and then we will try to get Dr. Young to commit himself on what was found in the kidney.

The chart shows that the temperature was constantly elevated throughout his whole stay here. He had never had a steady normal temperature from the time he entered until he died. It was very much higher in the last few days, but it reached 102° nearly every two hours for two weeks. So we ought to find something else besides cancer of the stomach.

DIFFERENTIAL DIAGNOSIS

I am not going back of the reasoning which Dr. Holmes brought before you in relation to cancer at the fundus of the stomach. He thinks it may possibly involve the esophagus too, but we have had no dysphagia or other swallowing symptoms. There is nothing in the rest of the examination that at all points away from cancer. There is an achylia. There are no gastric symptoms characteristic of gastric cancer, but any gastric symptoms that you can name are consistent with it. I do not think however that cancer is going to account for everything that Dr. Mallory is going to tell us. I think our chief business is to make some guesses as to what terminal infection the patient had. Could he have had a terminal infection in or about that cancer with a local peritonitis? I think not. There is not enough local disturbance in the way of spasm

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and pain, and there is nothing sudden in the onset of symptoms to warrant that diagnosis.

What are we to make from this streptococcus viridans? We suspect that he has an endocarditis. I do not see how we can rule out a vegetative endocarditis, a malignant endocarditis. He has also a systolic murmur. That is all he needs to have. It is enough to justify suspicion of that diagnosis. But we have no evidence of embolism unless these kidney symptoms may be taken as embolic.

Has he a renal suppuration, a suppurative nephritis, localized or diffuse, with pyelitis? I am not at all prepared to say that he has not. I can not make any better guess than that he has. I do not see anything else that is suggestive of other localization of inflammatory process except the heart (by the culture) and the kidney by the evidence that Dr. Young has just gone over with you. I think Dr. Mallory will not find a long-standing bacterial endocarditis, though I cannot exclude it. But something in the kidney, in the pelvis of the kidney, or in both seems on the whole more probable.

Dr. Young, would you like to commit yourself?

Dr. Young: I should like to say that when I was talking I did not get down to the streptococcus viridans. Of course that would mean a cortical lesion, and it is true that cortical lesions of the kidney may be present and very little may show in the examination of the urine. But that is not the common thing. There is generally persistent and marked tenderness in the costovertebral angle, and that generally increases with the onset of perinephritic infection. I wish they had taken a culture of the urine to tell us whether it was streptococcus there also. I believe, however, that Dr. Mallory will tell us that the kidneys are not infected as a primary cause of this condition, although I cannot say they may not be, any more than you can rule out the heart.

Dr. Cabot: You agree that we have to say something more than cancer of the stomach?

Dr. Young: I agree to that, and I think it is most likely to be one of those two things.

DR. CABOT'S DIAGNOSIS

Cancer of the stomach.
Acute endocarditis?
Suppurative nephritis or pyelitis or both.

DR. TRACY B. MALLORY
ANATOMIC DIAGNOSES

1. Primary disease.
Carcinoma of the stomach with perforation.
2. Secondary or terminal lesions.
Multiple polyps of the stomach.
Metastases to both adrenals.
Abscess adjacent to hilus of spleen.
3. Historical landmarks.
Chronic cystitis and pyelitis.
Cystitis cystica.

The primary cause of death in this case was, as diagnosed, a carcinoma of the stomach. The case was distinctly unusual in many other features, however. The stomach contained two small tumors of different character. One, occupying a large part of the greater curvature, was an ordinary benign

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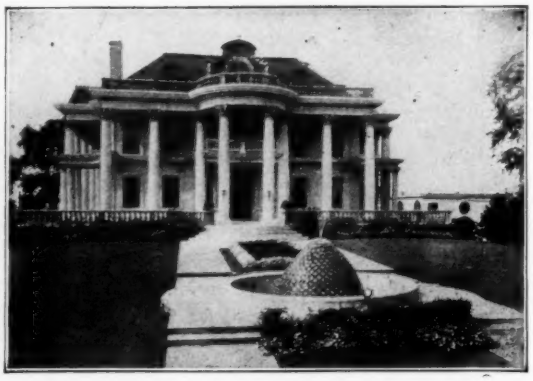

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polyp. The other tumor at its edges had the structure of a benign polyp, at the center the structure of a rapidly growing undifferentiated carcinoma. I think there is no question that cancer developed from one of the two polyps present in the stomach. If he had been caught a year or so previously they might have found only two benign polyps, I think. In the center of the carcinoma a small perforation had occurred and a small abscess was found lying between the fundus of the stomach and the hilus of the spleen. This was sharply localized. There was no generalized peritonitis. The tumor did not extend into the esophagus. It had invaded the left dome of the diaphragm over a wide area nearly ten centimeters in diameter, and large tumor nodules projected from the pleural surface of the diaphragm. I believe they might have shown in the x-ray plate if there had not been a moderate amount of fluid in the left chest.

The kidneys showed an apparently mild degree of pyeiltis with very little involvement of the renal parenchyma.

A slight degree of bronchponeumonia was present and probably accounted at least in part for the terminal rise in temperature.

Metastases were found only in the adrenal glands. Both glands were involved and apparently completely destroyed. No trace of adrenal parenchyma could be demonstrated at post mortem examination.

Dr. Cabot: It is very interesting, that last point. A man can have normal blood pressure and no evidence of Addison's disease when both adrenals are diseased. It is not the first time I have heard it, but it always surprises me afresh. I suppose it means that the essential part of the organ is still intact.

We had three infections at the end, the abscess and perforation of the cancer in the stomach and spleen, the mild pyelitis and the bronchopneumonia.

—A Baker Institute "Diagnosis."—The history of every quack concern that professes to cure cancer is monotonously alike. The scheme consists in diagnosing every simple skin lesion, no matter how benign, as cancer. Caustics are then applied and a hole eaten in the tissues with inevitable disfigurement and the patient finally sent back home "cured." Of course occasionally the quacks get real cases of malignant disease. Most of these are sent back home in time to avoid the necessity of the "institute" having to sign the death certificate. A recent case is of interest in that the victim showed more intelligence than is frequently displayed and thus saved himself considerable suffering and disfigurement. Mr. I., an Iowa farmer, developed a lesion on the chin that worried him. He went to Muscatine to the Baker Institute where he was "examined." Mr. I. reports that he was told that he had cancer of the chin and would have to pay two hundred and fifty dollars and also sixty dollars a week hospital charges for four to six weeks. The Baker Institute, according to the victim, applied their cancer remedy. Then the young man got to thinking and decided that the Iowa State University was not far away and that they probably knew as much about cancer as Mr. Baker and his "institute." He went to the College of Medicine of the state University of Iowa where the dermatologic department diagnosed the lesion as **Tinea barbae.** Treatment for ringworm of the beard was instituted and in less than two weeks the young man went home. (Jour. A. M. A., July 26, 1930, p. 285.)

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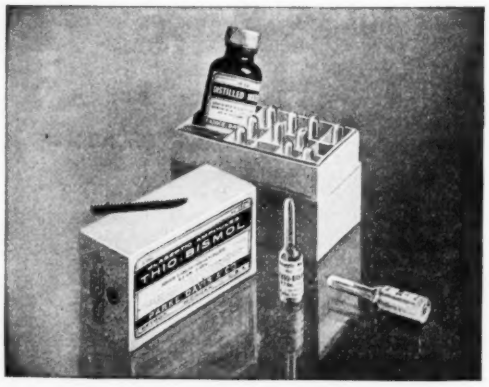

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